

905

# THE BOSTON Medical and Surgical JOURNAL

VOLUME 191

OCTOBER 30, 1924

NUMBER 18

## ORIGINAL ARTICLES

### SOME PRACTICAL POINTS IN THE CONSIDERATION OF DISEASE OF THE UPPER RIGHT ABDOMEN\*

BY JOHN B. DEEVER, M. D., PHILADELPHIA, PA.

I MAKE NO apology for the choice of my subject except to say that I thought it might be of interest both to the internists and the surgeons present to discuss some of the most important pathologic conditions arising within the upper right abdomen, which come within the experience of all of us. In the order of their frequency these would be an appendix in a high position (for the appendix must necessarily be included in disease of the upper abdomen), infection of the biliary tract, including the pancreas, acute and subacute perforated ulcer and subdiaphragmatic abscess.

The appendix has been justly accused of being able to mimic almost any of the diseases of the upper right abdomen. This is in some respects due, as you know, to anatomic variations in its position. The position naturally influences the direction of the pain. When the organ points upward the pain will be referred in that direction; if it points downward the pain is referred to the pelvis and if to the left there will be pain referred to the left side. In fact, the pain in appendicitis may be almost anywhere within the abdomen, and might even extend to the calf of the leg, if the appendix were long enough to pass through either sacrosciatic foramen and extend beneath the deep fascia of the thigh and the leg. It is the appendix in a high position that is our concern at this time.

Oftentimes the diseased appendix in such a position can so exactly simulate symptoms of gall bladder trouble that it is next to impossible to make a clinical diagnosis even with the aid of a well-taken history and x-ray study. Indeed, the appendix is sometimes found wrapped around the cecum directed upwards and reaching almost to the gall bladder so that the end of the appendix, the duodenum, the pylorus and the head of the pancreas are all grouped together. In such a case the clinical symptoms may be those of cholecystitis, chronic pancreatitis or ulcer with pylorospasm, or both, while the appendix acts as a silent partner furnishing the wherewithal for carrying on. In a case of acute fulminating appendicitis when the organ occupies a high position and it perforates at the

base there is the same boardlike rigidity and other symptoms and signs closely resembling those of an acute perforating duodenal or gastric ulcer. I occasionally have been misled in such a case, having made a diagnosis of perforating ulcer only to find a perforation of the base of the appendix. Happily this does not occur often, for if it did the mortality of appendectomy for acute appendicitis would be much higher than it is. The differential points are mainly in the history. Appendicitis, for example, generally gives a history of previous attacks of short duration yielding to rest, the application of icebags and abstinence from food. There is local discomfort between attacks aggravated by exercise, and tenderness to light pressure at the site of the appendix with slight rigidity of the overlying abdominal walls.

The chief differential point in the history of an acute fulminating appendicitis with perforation at the base and acute perforated duodenal or gastric ulcer is that in the former there will be elicited the history of at first general abdominal pain, not very severe, but cramp-like and referred to the epigastric or umbilical region, soon followed by very severe pain and boardlike rigidity of the muscles. In the ulcer patient there will be simply the history of severe pain coming on like lightning out of a clear sky, followed immediately by the characteristic muscular rigidity which once seen is never forgotten. The acute perforated ulcer patient does not always give the history of ulcer, while the appendix patient is more likely to give a history of previous trouble. Why? Because ninety per cent. of patients with a perforated appendix have had one or more previous attacks. Here let me pause to say that these cases of perforated appendix usually have been sick long enough before perforation occurs for a purgative to have been inadvisedly given, which may have precipitated the perforation, while in acute perforated ulcer there is no warning of the oncoming of the perforation.

In an ulcer patient where the previous history is that of ulcer, the diagnosis is perhaps made with more confidence, but we must keep in mind the atypical ulcer, the ulcer that does not present the so-called characteristic ulcer his-

\*Read before the Berkshire District Medical Society, Lenox, Mass., July 31, 1924.

tory. To repeat, a sudden attack of most agonizing abdominal pain immediately followed by boardlike rigidity of the abdominal walls means a perforated ulcer in ninety per cent. of cases. In the perforated ulcer cases coming into the Lankenau Clinic the diagnosis is made by my internes, who call me on the 'phone saying, an acute perforated ulcer case has been admitted, and the diagnosis is practically always the correct as proven by immediate operation, with the exception of the occasional perforated appendix, to which I have referred; or very rarely a ruptured gall bladder or mesenteric thrombosis. In this connection I wish to say that the operation that will accomplish the best immediate and remote results in the acute perforated ulcer is closure of the perforation and a posterior gastroenterostomy. This was my practice formerly, but recently for a time, when the perforation was small and the surrounding exudation slight, I stopped doing a gastroenterostomy contenting myself with simple closure. My reason for getting away from my original practice was that the consensus of surgical opinion was against the procedure. However, my later experience has convinced me that I was wrong in changing, and I have again resumed the practice.

When a hole in either the duodenum or the stomach is closed by suture, and reinforced by a piece of omentum, the risk of leakage, while it is minimized especially when there has been but little if any induration around the hole, is greater than when a gastroenterostomy is also done. I have at times seen leakage occur where only the opening was closed, where there was considerable induration, therefore, if one is going to close only the opening made by the perforation, it is better to cut or burn out the induration if the latter is not too extensive and then close it, thus securing better and more secure apposition of the tissues. This is not feasible when the induration is fairly wide spread and especially if organization has advanced to any degree and the location of the opening is difficult to get at. A gastroenterostomy provides rest for the sutured area, relieves tension of the tissues approximated in providing drainage for the stomach, and renders the future of the patient safer against ulcer re-formation. In short, in the early cases of perforation greater good is accomplished by this method of operating. The late cases, with peritonitis already advanced, promise but little with any operation, but more perhaps when the perforation alone is closed. I have seen so many recoveries follow the method I have here advocated that I strongly declare in its favor, notwithstanding the objections offered by many.

In the small chronic, non-perforated ulcer, excision alone may suffice, but as in the acute perforated ulcer the addition of a posterior gastroenterostomy is of advantage.

In subacute perforated ulcer there is the usual history of indigestion with discomfort or perhaps actual pain after eating. The history is a prolonged one with attacks at certain periods and well being between attacks, and finally a comparatively acute exacerbation of pain at the time of perforation followed by a circumscribed peritonitis resulting in more or less permanent invalidism consisting of continual soreness in the upper abdomen, constant distress after meals with rigidity of the overlying muscles and tenderness certainly to deep if not to moderate pressure. These cases of subacute perforated ulcer are deceptive, and in the absence of a clear ulcer history are often mistaken for a chronic gall bladder, chronic pancreatic affection or a chronically diseased high lying appendix. It matters but little if the correct diagnosis is not made so long as there is pathology responsible for symptoms that cannot be relieved by means other than operation.

Biliary tract infection most commonly attacks the gall bladder resulting usually in two types of cholecystitis, one attacking the mucosa and the other the walls of the organ. Mucous or catarrhal cholecystitis comprises the cases which after an acute attack, in the absence of gallstones, usually subside without showing further trouble. But the interstitial infection always leaves some residue of the inflammatory process which sooner or later manifests itself in the shape of recrudescence of attacks together with the development of gallstones and gradually involvement of the pancreatic lymph glands and the pancreas itself.

Involvement of the pancreatic lymph glands produces no clinical symptoms and can be demonstrated only at operation. The careful observer can see the process often enough to be convinced of the presence of the condition as the direct result of biliary tract infection and a forerunner of the more serious condition of chronic pancreatitis. As for the symptoms of the latter they are not as yet definitely formulated mainly because, as a rule, they are so intimately associated with a co-existing cholecystitis, and even when at operation the gall bladder appears normal the clinical symptoms in the case will bear a strong resemblance to a gall bladder history. It is therefore no exaggeration to say that uncomplicated chronic pancreatitis can be diagnosed in most instances only by inspection and palpation of the exposed organ. Generally speaking the syndrome is somewhat as follows:—There is marked indigestion, fullness and belching of gas immediately after eating, tightness or distention in the upper abdomen, occasional paroxysmal pain in the epigastrium or the right hypochondrium sometimes referred to the lumbar region. The pain may radiate to the right shoulder, the back and the left shoulder or to the lower abdomen, with the frequency in the order named. Vomiting is somewhat more frequent than in other chronic diges-

tive disorders. Jaundice is not characteristic of pancreatitis *per se*. It is sometimes present together with clay colored stools and bile in the blood serum and urine. Obstruction to the flow of bile, however, is never complete in these cases, so that a small amount of bile may reach the intestine and be demonstrable in the stool as stercobilin. But jaundice with pancreatitis in the majority of cases is due to gallstones which so frequently complicate the disease, or to cholangitis or pericholangitis. The supposedly typical large, bulky, greasy stools are seldom seen and when present suggest an irremediable condition.

As to the various tests, including duodenal tubage, I cannot say that I find them of much if any value. Our experience in the Lankenau Clinic with these tests made for the purpose of determining the operability of the case have been most unsatisfactory, so much so that we now seldom use them. This is borne out in operations where they have been made before our seeing the patient, inasmuch as the operative and bacteriological findings made from the bile aspirated directly from the gall bladder and common duct at operation does not agree with the pre-operative findings. Duodenal tubage gives only temporary relief. This is the information we have gotten from the many patients we have operated upon. Moreover I believe the practice is not entirely free from the risk of contaminating the pancreas through the duct of Wirsung.

If we are correct in believing that most of the infections of the biliary tract originate in the liver, and that the biliary passages, including of course the gall bladder and pancreas, become infected secondarily nothing other than wide open drainage is logical. This has been borne out in my experience in the many bile tract drainages both by tube and cholecysto-duodenostomy that I have made. Our Follow-Up System in our Clinic, now in operation four years, confirms this statement. Furthermore these many living autopsies with the bacteriological and pathological examinations that go hand in hand with our operations have left no doubt as to the facts. We are constantly operating cases that have had the Sippey treatment for ulcer, the Lyons and Rehffuss treatment for gall bladder disease, where only a chronically diseased appendix is found. Undoubtedly the most useful of the laboratory methods is the x-ray and the fluoroscope, yet these are far from being infallible, and while they should be used, should not be wholly depended upon. The clinical side still holds the most important place. What can be learned by the refined use of our special senses plus common sense, backed up by a long experience still constitutes art.

Jaundice in biliary tract infection is intense only in the presence of calculous and neoplastic obstruction, and occasionally of a cholangitis and peri-cholangitis. The latter, as already indi-

cated, is the cause of the jaundice seen in cholecystitis and pancreatitis. In cholecystitis, if the jaundice has not preceded the gall bladder infection, it is due to extension of the inflammation into the hepatic duct and its branches, while in pancreatitis it is more likely due to pressure upon the terminal part of the common duct. Relief of this variety of jaundice is only accomplished by drainage either of the gall bladder, or of the common duct or by a cholecysto-duodenostomy. It is understood that to secure drainage through the gall bladder the organ must be comparatively free from disease and the cystic duct patulous. Drainage by means of a cholecysto-duodenostomy should be ideal, but we cannot tell how long the cholecysto-duodenostomy stoma remains patulous. In re-operating patients where this operation had been done (elsewhere) several months previously, I have at times found the opening completely closed. This has not occurred in any of my own cases so far as I know. It is my practice to make the stoma as large as the size of the gall bladder will permit. When the gall bladder is unusually small the operation is not feasible. I do not make a cholecysto-gastrostomy, as is the practice of many surgeons. I do not consider anastomosis of the gall bladder to the stomach as effectual as the anastomosis of the gall bladder to the duodenum. The latter procedure is a physiological one in that it deposits the bile where it naturally empties. I do not find the operation difficult, especially when the second portion of the duodenum is mobilized by dividing the peritoneum along the extreme postero-lateral wall of the duodenum. Up to the present I have not seen leakage, (a fistula,) follow the operation in any of my cases. It goes without saying the technique requires care, that is, one must cut well and sew well in order to have the patient get well. In a number of cases of diffuse interstitial pancreatitis I have not only drained the common bile duct by introducing a rubber T-tube but have made a cholecysto-duodenostomy as well. This will impress you with my confidence in the operation of cholecysto-duodenostomy. Drainage to accomplish a cure in chronic pancreatitis must be prolonged. For example: when I drain the common duct under these conditions I rarely remove the tube in less than six months and sometimes have the patient wear it for one to one and a half years. I had one patient who wore a T-tube for four years. This was a woman who suffered from a chronic cholangitis and who had had two previous drainage operations of short duration. At the third operation with the T-tube introduced she remarked to me that this tube would remain for an indefinite time as she did not want any more recurrences of the trouble. This particular patient suffered from jaundice, chills and profuse sweats, but no pain; she also suffered from annoying itching. When I saw her in consultation before the third operation and asked her

if she perspired much she told me she saturates six night gowns every night.

The cases requiring prolonged drainage as a rule have suffered for a long time, during which time much medication has been used, numerous duodenal tubages made, many x-ray pictures taken, and in not a few instances many trips have been made to one or other of our famous springs and to hydro-therapeutic and helio-therapeutic institutions, etc.

But to get well, I say, they must be drained. My relation to these patients is much like that of a famous undertaker in Philadelphia who has erected a million dollar funeral establishment, of whom it is said his favorite expression was, "You may linger but I will get you by and by." So I can say I get these inviolable chronic cases of pancreatitis by and by after they have gone the rounds of the internists and the gastro-enterologists who work by faith and not by sight. The inference to be drawn from these crude remarks is that patients with biliary tract disease should be treated surgically early in the disease and not late, if the best as well as the most permanent results are to be obtained. The surgeon can never be called too early but can be called too late. Explanation of the rationale of cure by drainage is that the patients get well. There is no other way I know of to get them well. However before resorting to an operation in an early case, all foci of infection must be sought for and removed. Particularly should the alimentary canal receive the closest attention, especially the colon.

A condition of the right upper abdomen that I see many times during the year is subdiaphragmatic abscess, often difficult but not impossible to diagnose. The history, with careful physical examination, which should include not only the site of the lesion but the chest as well, will often suffice to reach a conclusion. Inspection, percussion and palpation should be carefully done. Palpation will usually elicit tenderness significant of the location of the collection. I place much reliance upon marked tenderness to deep finger pressure, particularly when attempting to differentiate between a subhepatic and a suprahepatic (subdiaphragmatic) collection. This examination with the judicious use of the aspirating needle, the x-ray and fluoroscope are our best means of reaching a correct conclusion. A collection beneath the liver may occasion some doubt, yet if one palpates, percusses and auscults carefully, it can be determined. The aspirating needle should never be used here. It is wiser, safer and surer to incise than to aspirate. In our experience the most common cause of subdiaphragmatic abscess is acute suppurative appendicitis.

Visceroptosis, colitis, &c. are fruitful sources of upper abdominal lesions along with appendicitis. Treatment by colonic irrigation properly done is of advantage, and in a few instances autogenous vaccines, after careful bacteriological

studies and the isolation of the particular micro-organism, may do good. We are well aware how prone some of these sufferers are to develop hypertrophic arthritis, therefore it behooves us to be real doctors ever watchful and careful in our investigation of our cases in order to reach logical conclusions.

Incidentally I may say I do not advocate radical surgery for viscerptosis and colitis, which as you know are always accompanied by more or less marked stasis and usually by a large nervous element. Surgery is uncertain in this type and from the standpoint of discretion I hesitate to launch into uncertain fields, since the labor in too many instances is in vain and may leave the patient worse off. This is an age of adventure and the spirit of it has not passed the surgeon by. We hear much of the accomplishments of new operative procedures, for example: peri-arterial sympathectomy for the cure of terminal endarteritis in the shape of senile gangrene, cervical sympathectomy for the relief of angina pectoris, the much lauded cures by radium, the cure of cancer by serum, &c. To all of these I give a deafening ear. I may be wrong, but my experience has told me to—Stop, Look and Listen. This vividly recalls to my mind a criticism made by the late Dr. Agnew of the operation of craniotomy for the cure of idiots, when he remarked, he could not see the philosophy of enlarging the shell of a nut the kernel of which was dead. I cannot see the philosophy of operating for conditions such as terminal endarteritis, aortitis, or sclerosis of the coronary arteries by nerve section which does not attack the pathologic source of the evil. And so I could go on putting up arguments against procedures not based upon a knowledge of fundamentals.

#### DISCUSSION OF DR. DEAVER'S PAPER AT LENOX:

DR. FRANK H. LAHEY

It has been exceedingly interesting and instructive to hear Dr. Deaver's paper and particularly entertaining to listen to his remarks aside from his paper. It is, of course, impossible for me to disagree with what Dr. Deaver has said and I would therefore like only to emphasize one or two points which he has brought out and to mention one or two things as the result of our own experience in this field.

The first is subdiaphragmatic abscess. This condition is often the cause of persistent fever after right-sided septic intraabdominal processes, and from our experience we believe that it occurs more often than is commonly thought. Since one of the staff of our clinic, Dr. H. M. Clute, who has been particularly interested in this subject for some time, has been on the lookout for these cases, we have found altogether nine cases, a considerably larger number than had hitherto been discovered. In this group of cases it should be recalled that subdiaphragmatic accumulations elevate the diaphragm



without pushing the liver downward; that at times there is a bubble of gas above the pus which shows as a transverse straight fluid line in the X-Ray. This phenomenon, unfortunately, is only occasionally present. Oedema over the chest wall, with, at times, obliteration of the intercostal spaces occurs. In all cases with persisting temperature following septic abdominal lesions, X-Ray examination of the diaphragm should be made.

In needling suspected cases of subdiaphragmatic abscess it is desirable that facilities for immediate operation be at hand, since the exploring needle must be introduced at the level of the tenth rib where, if subdiaphragmatic abscess exists, the diaphragm is so elevated that the needle reaches the pus beneath it only by passing through the parietal pleura, traversing the costo-diaphragmatic angle and then penetrating the diaphragmatic pleura and diaphragm itself. If pus is obtained, immediate operation by the two-stage plan should at once be undertaken, the tenth rib removed, the parietal pleura pushed away and fixed to the adjacent elevated diaphragmatic pleura, thus shutting off the pleural cavity and protecting it from infection. If, on the other hand, hours are allowed to elapse between the needling and withdrawal of pus, a sufficient amount of this material may escape through the needle-hole in the diaphragm into the pleural cavity to infect that cavity and produce an empyema.

During past years I have several times done exploratory operations upon cases of persistent, progressive and painless jaundice only to find, as is so constantly the case in jaundice of this type, either a carcinoma of the head of the pancreas or of the ducts themselves. With cases of this type I must admit to having had a certain degree of guilty feeling, so regularly has the obstructive jaundice proven to be of malignant origin, and up to the past year or so I had almost come to feel that exploration in cases of this type entailed needless suffering without adequate possibility of cure. During the past year, however, we explored such a case of typical obstructive painless jaundice of four months standing, finding a stone at the ampulla of Vater. In view of this case with complete absence of pain and the relief from removal of the stone, I shall again continue to feel that exploration in this type of case is entirely justifiable even though an overwhelming majority prove malignant in origin and inoperable in character.

There is an unusual but not rare complication of gall bladder, bile duct and pancreatic infections which is most difficult and in the past has been most unsatisfactory to deal with. I refer to those cases in which the common duct is so injured at the time of operation or as the result of the infectious process that a persistent biliary fistula is established, all bile draining onto the

abdominal wall and none reaching the intestinal canal whatever.

Up to the present the methods advocated for restoring the flow of bile to the duodenum have been to find the intact end of the common or hepatic duct, to insert a small piece of rubber tube into the end of the duct, the other end being inserted into the duodenum, and the tube then wrapped in omentum in the hope that a biliary tract would be established when the tube was passed into the intestinal canal. Attempts have been made also to construct a duct from a flap of gastric wall and also the cut end of the duct has been found and attached to a loop of adjacent bowel. A great majority of these cases have been failures due to the technical difficulties of the procedures in the latter two methods and to closure or infection of the canal in the first method. Last year I published in the *Journal of the American Medical Association* a method of preserving the fistulous tract running to the abdominal wall and anastomosing it to the duodenum, and reported a case of three months' standing in which I had successfully made the anastomosis with healing, without leakage, and the establishment of bile-colored stools. Since then I have had two other cases sent to me and have successfully transplanted the complete biliary fistulae with the establishment of bile stools. One of the cases proved to have carcinoma of the head of the pancreas, from which he died a few weeks after operation, but with no evidence of jaundice. In the last case the fistula had to be anastomosed to the pyloric end of the stomach, because the adhesions subsequent to the original cholecystectomy were of such character as to prevent the approximation of the fistula to the duodenum.

If one recalls that external biliary fistulae remain open indefinitely provided the patient does not die of inanition (the last case had a complete biliary fistula with acholic stools for twenty months before the anastomosis was made), it must be accepted that the force of the secretory pressure of bile is greater than the ingrowing force of the cicatricial tissue in the fistula walls. In my original operation I therefore conceived this procedure by assuming that if the secretory pressure of the bile was sufficient to maintain an external biliary fistula, it should be sufficient to maintain an internal one. In reporting this method I ascertained that Dr. Hugh Williams had done a similar operation successfully nine years ago, but had not published any report of it. Priority for the procedure, therefore, is properly his.

It is a comparatively simple operation, much easier to perform than any of the other proposed procedures, much less liable to leak, I believe, and much more liable to maintain the patency of its canal. I recommend it to you as a possible measure in the treatment of a condition hitherto almost hopeless in outlook.

## CONGENITAL STENOSIS OF THE PYLORUS

## Fredet-Rammstedt Operation on the Stomach of a Four Pound Infant Under Novocain Anesthesia

BY WILLIAM REID MORRISON, M. D., F. A. C. S.

*Assistant in Anatomy, Harvard Medical School; Instructor in Surgery, Tufts Medical School; Junior Visiting Surgeon, Boston City Hospital*

A MOST important and serious gastric condition, which is comparatively rare, in young infants, is a peripyloric obstruction at the outlet of the stomach, known as congenital hyperplasia of the pylorus. There is more or less associated edema of the mucosa and submucosa, resulting in an intrapyloric stenosis, or blocking of the lumen of the bowel.

This pathological situation, in complete or nearly complete obstruction of the pylorus, demands surgical intervention just as urgently as does an acute partial enterocoele, or a beginning strangulation of the bowel, so commonly seen in hernias, or due to contractions of ulcers or carcinomas of the intestinal tract. The general practitioner, who usually sees this condition at first hand, now as a rule makes an early diagnosis, confirmed at operation or autopsy.

In the future, in cases of complete or almost total obstruction, the mortality will be greatly lowered, if early surgical intervention is advised by the family doctor, or internist, and allowed by the parents. Appropriate pre- and post-operative treatment is very essential for success.

## DEGREES OF PYLORIC OBSTRUCTION

Infants may have mild, moderate, or severe symptoms of stenosis at the pylorus; the evidences of trouble may be periodic, with remissions, or progressive in character. It seems to be the general opinion of modern writers that the mild or moderate syndrome, as observed in the so-called pylorospasm cases, is an evidence of mild or moderate obstruction. In such cases, medical treatment should be carefully given, allowing a fair trial to the various formulae, including the thick cereal feedings, and small doses of atropine. This should be carried out preferably in consultation with a skilled pediatrician, for the benefit of all concerned. After all, it is a matter of common sense and good judgment when to interfere surgically.

## WHEN TO OPERATE

Strauss believes that in any case in which one-half or more of the barium mixture remains in the stomach at the end of four hours, the patient should be operated on.

Downes states that surgical intervention is indicated in every case in which definite obstruction is present, or seems imminent. Furthermore, he thinks that any patient under medical care, suffering a relapse, should be operated on

at once, and that all cases of ten days' standing, or longer, in which there are no data as to weight, and the patient is not in good condition, should be surgical. He thinks that medical treatment is justified for seven to ten days, if the baby does not lose more than twenty per cent of its body weight. The smallest baby he has operated on weighed three pounds, fifteen ounces.

Tyrrell and Reynolds declare that a decline in weight clearly constitutes an urgent indication for surgery, especially when near the border line of six pounds. They think that prolonged medical treatment is responsible, with ether anesthesia, for a large proportion of fatal cases.

Haggard writes that only very mild cases should be treated medically and tentatively; all others should be operated on. In mild cases with little hyperplasia, the infant may outgrow the condition causing the symptoms.

Palmer reports that an operation is safer than the care of a poor pediatrician, even though the symptoms are mild; it is also safer than the average unintelligent treatment by the parents. He considers the child's condition and need of operation, by the relation of the present age and present weight to the birth weight. He believes that a border-line case should be surgical, and that a well developed case must be surgical.

Porter observes that medical treatment causes children to be invalids for long periods of time, when intercurrent diseases are exceptionally fatal, on account of lowered resistance.

Tumperer and Bernstein believe that the Fredet-Rammstedt operation corrects both the anatomic and nervous conditions present in stenosis, by splitting the tumor and interfering with the nerve supply of the region.

## OCCURRENCE

According to Hill, five out of every thousand babies suffer from this malady. It is often difficult to determine exactly the presence or absence of stenosis, or its degree, except at operation or autopsy. Scudder has pointed out that formerly death certificates were signed inanition, acute gastritis, infantile atrophy, gastrointestinal catarrh, dyspepsia, or pyloric spasm, in the absence of operation or autopsy.

This disease is more common in males than in females, in the ratio of seven or eight to one, according to Wall; the usual estimate is not so large.

Tarr states that it occurs in breast fed infants about as often as in artificially fed babies

CAUSE

Wollstein has shown that the lesion in pyloric stenosis is a hyperplasia of the unstriated muscle cells of the circular coat, while the connective tissue is not increased. There are several views as to the cause of the hyperplasia: (1) congenital malformation, a primary developmental hypertrophy, (2) spasm and hypermotility of the pyloric musculature, (3) incoordination between the movements of the stomach and pyloric walls, (4) hyperadrenalism, the result of excessive sympathetic stimulation. A congenital anomaly is the most generally accepted cause of most writers. As evidence for this belief, Wall has pointed out that there is a temporary obstruction of the lumen of the duodenum, during the normal development of a fetus. In embryos of 12-13 mm., the lumen of the duodenum becomes obliterated by an overgrowth of the mucous membrane caudal to the ducts of the liver and pancreas. In embryos of about 15 mm., the lumen reappears. In a 22.8 mm. embryo, a model was made by Johnson at the Harvard Medical School, showing that the passage from the stomach to the jejunum was completely blocked by epithelial septa. At 30 mm., the vacuoles begin to become confluent so that a central lumen is re-established. Tandler in 1900 was the first to recognize that the duodenal lumen in embryos from 30-60 days is normally more or less obliterated.

Well-marked tumors of pyloric hypertrophy have been reported in the new-born, as well as in premature infants; Dent found a tumor in a seven-months fetus.

Scudder has called attention to the fact that pyloric tumors are associated occasionally with other congenital defects, as imperforate anus and club-foot. Palmer states that an unduly high percentage of cases of thymus enlargement are associated with stenosis, but thymus enlargement occurs in a large per cent of normal children.

Downes thinks that there is a true malformation present at birth, consisting of an abnormal thickening of the circular muscle of the pylorus, and the effort to force food through the narrowed and elongated pyloric lumen produces circulatory disturbances resulting in edema. By the tenth day, the lumen is more or less completely obliterated, spasm being a superadded result. Any one who has seen and felt these tumors of cartilaginous hardness could hardly ascribe their cause to muscle spasm alone.

Strauss tried experimentally, to develop pyloric tumors in new-born puppies, with negative results. The mucosa was shelled out, and infolded with fine fascial strips, but no tumor was produced. Fine platinum electrodes on the tenth nerves increased peristalsis, but did not cause any tumor to form. He believes that the hypertrophy may be due to an abnormal stimu-

lation from the intrinsic or extrinsic nerves of the stomach. Tumperer injected paraffin, 8-15 cc., into the muscularis of the pylorus of dogs, producing the anatomical conditions seen in stenosis.

Pyloric tumors at 7-12 weeks of age are usually four or five times as large as those at 3-4 weeks, but the size of the tumor does not indicate the amount of obstruction. Tumors relatively as large as the usual twelve weeks old size may appear at 3-4 weeks. Maylard reported a case in a woman of thirty-seven, probably of congenital origin.

Thomson has pointed out that the size of the pylorus increases very greatly during the early weeks of life, and the mucous membrane is thrown into folds. Similar folds have often been found in the posterior urethra in cases of congenital hypertrophy of the bladder with hydronephrosis; a condition somewhat analogous to congenital pyloric hypertrophy. Young, Frontz, and Baldwin reported twelve cases from Johns Hopkins in 1913. There is no hypertrophy around the valves in the urethra, according to Stone.

Tyrrell and Reynolds observe that the closure of the sphincter is controlled by stimulation of the sympathetic nerve supply, and by its hormone adrenalin, therefore pyloric hyperplasia is associated with hyperadrenalism, the result of excessive sympathetic stimulation. They believe that hyperadrenalism is maternal, caused by a natural mother's anxiety, accentuated by first children, children born after a long interval, children born while the husband's life was in jeopardy, children born when all the other children died in infancy. They note also that phimosis, adherent prepuce, retained smegma, and balanitis may all provide a focus of constant irritation, which might cause excessive nerve stimulation. This of course is pure theory.

SYMPTOMS

A careful history is most essential. There is at first a stage of the infant's life without symptoms, anywhere from one to eight weeks after birth, usually two to four weeks. This is a latent period of well being after birth. Later, there is a loss of appetite, followed by discomfort, then persistent, recurrent, explosive vomiting, often described as projectile, or geyser-like. The vomiting depends on the quantity, not the quality or kind of food; it also happens directly after feeding, unless there is dilatation of the stomach, then collective vomiting occurs. The vomitus does not contain bile, in complete obstruction at the stomach outlet. Allen states that absence of bile is not pathognomonic of this disease. Wall observed that the vomiting occurs usually between the third and sixth week of life, and it is usually in breast-fed babies, because month-old babies are usually breast fed.

The vomiting rarely occurs before the tenth day or beyond the sixth week. It is not regurgitation; the vomiting is large in amount; the time of vomiting is usually during the act of nursing or shortly after. It may become forcible, and is shot across the room four or five feet; it is the typical vomiting of obstruction at the outlet of the stomach. A change of food for a day or so may abate the vomiting.

Haggard states that the next most important symptom is a starvation or hunger stool, with diminution of urine and emaciation. The stool contains mostly bile, pancreatic juice, and epithelium from the intestines, and appears like meconium. There may be prolonged retention in the stomach of food which is not vomited. There is marked drying up of all the soft tissues, with no gain in weight.

Visible, characteristic gastric peristalsis, with epigastric fullness and stretched abdomen, and a definite loss of weight follow, with acidosis.

A pyloric tumor is always present in real congenital hyperplasia with stenosis. Inability to discover a tumor by palpation does not exclude a positive diagnosis, with other characteristic symptoms, and in a child not over six weeks old. It is white in color, and is usually described as olive shaped, and movable, situated in the epigastric region. It is about the size of the terminal phalanx of an adult thumb or finger, oval, firm, smooth, and movable. The tumor has been described as spool-shaped, like a pea, hazel-nut, hickory nut, pecan, or peanut, or lymphatic gland, and often as hard as cartilage. Haggard described, at autopsy, a napkin-ring-like tumor projecting into the duodenum. The shape of the duodenal opening of the infantile pylorus has been likened to the primiparous cervix in the vagina. I recently saw a case with a tumor the size of a small marble, which was difficult to feel, as it was hidden by the left lobe of the liver, and at operation the tumor was the size of a small olive, and hard as cartilage. The transverse diameter was felt through the abdominal wall, because the long axis of the bowel pointed toward the back, under the liver. The tumor is usually described as being an inch to the right of the median line, and midway between the ensiform and the umbilicus. The tumor can be felt best in the brief period of relaxation following vomiting.

There are never any adhesions about the tumor, which has a sharply defined outline, and is covered with smooth glistening peritoneum.

Palmer considers the presence of the pyloric tumor the least important of the symptoms. The tumor may be mistaken for an enlarged lymphatic gland, or the lower pole of the right kidney.

Above the pyloric obstruction, the stomach wall may be thicker, but usually is thinner than normal, the esophagus and the stomach may be dilated, while below the stenosis, the intestines

are collapsed and empty. Downes recommends emptying the stomach of gas by passing a small catheter, before palpating the abdomen. Relaxation of the abdominal walls is favored by letting the baby suck water from a bottle. Ether or ethyl chlorid inhalation may be used to insure absolute relaxation of the abdominal muscles. Flicking the abdomen with a towel, or snapping with a finger produces peristalsis.

The progressive loss of weight, in serious cases, causes great emaciation, loss of elasticity of the skin, and debility, with symptoms of toxemia, such as flatulence, colic, diarrhea, drowsiness, and sometimes convulsions. Obstinate constipation is more often present than diarrhea. Shrunken, wizened body, sunken fontanelles, concentrated or suppressed urine, and meconium-like stools without food content, are danger signals. The child at first is very hungry, but in the later stages of obstruction there is a lessened desire to take nourishment.

Downes states that most stomachs in these cases are large, but some are small, holding only one ounce. Edema, varying in degree, involving the pylorus and pyloric region of the stomach, was present in all his cases. The extent of the edema may cause marked variations in the severity of the symptoms.

#### DIAGNOSIS

Strauss believes that fluoroscopic examination is the most important means of diagnosis. He does not undress the baby; he adds bismuth to the mother's milk, and under a horizontal fluoroscope, observes the stomach while the baby nurses from a bottle. The baby should not lie on its back, for the bismuth-milk gathers on the left side of the vertebral column, as a large round mass; it will stay there indefinitely, and no peristaltic waves are stimulated at the pylorus. The baby should be rotated to the right side, and characteristic snake-like, rhythmic, peristaltic contractions appear in the pylorus, which are independent of the rest of the stomach. This is absolutely pathognomonic of congenital stenosis, according to Strauss. The fluoroscopic examination should be repeated at the end of two hours; then after four hours, x-ray plates may be taken. Retroperistalsis may be seen in the body of the stomach, and in a dilated esophagus. The rounded shape of the pyloric end of the stomach is of significance in the x-ray as evidence of obstruction.

Palmer believes that the gross gastric wave is the most important single symptom, since it is always present, or has been present; he points out that in extreme emaciation, atony and dilatation of the stomach may prevent signs of peristalsis, visible through the abdominal wall as seen by means of the fluoroscope. He observes that the waves are usually accompanied by abdominal pain, and are most active preceding



the projectile vomiting. Peristalsis from right to left is particularly significant.

Holt bases a diagnosis on (1) history, (2) abnormal gastric retention, (3) peristaltic gastric waves, (4) palpable tumor, (5) wasting, constipation, and scanty urine.

Haggard gives the following symptoms in the order of their importance: (1) persistent, recurrent, explosive vomiting; (2) starvation stools, and diminished urine, with emaciation; (3) visible gastric peristalsis; (4) palpable tumor; (5) bulging epigastrium with stretched abdomen; (6) progressive loss of weight.

According to Curtis, the type of feeding is a chief diagnostic point, spasm being extremely rare in the breast fed. He thinks that if a breast-fed baby shows symptoms of spasm or tumor, the chances are greatly in favor of a real tumor being present.

Symptoms may appear within ten days after birth, or be delayed until the second or rarely third month; usually, according to Haggard, they appear during the third or fourth week. It is a fact that hypertrophic stenosis may, in rare instances, exist for years before it causes symptoms. Rosenheim reported a case of a boy having no gastric symptoms until over five years of age. At operation, at six and one-half years, a stenosis was found at the pylorus.

Thomson divides the main clinical features into four stages: (1) stage without symptoms, one to eight weeks after birth, usually two to four; (2) stage of primary symptoms, having characteristic violent vomiting, prolonged retention in the stomach of food not vomited, drying of all the tissues, no gain in weight, and visible gastric peristalsis; (3) stage of secondary symptoms, denoted by emaciation, debility, dilatation of the stomach, mucus in stomach contents and feces, symptoms of dyspepsia and toxemia; (4) if the child survives, as is the case in partial stenosis, period of recovery. He believes the disease to be self-limited, and if the child does not die of inanition from too little food reaching the bowel, natural processes of growth and development will, in time, remove the obstruction. However, the pylorus has been shown to be closed, by x-ray examination, eight or nine years after a gastroenterostomy for the disease.

Probably the hyperplastic pyloric stenosis occurring in young adults may be explained on the basis of a spontaneous cure during infancy, with the hyperplasia remaining, according to Haggard.

Repeated observations should be made, with the infant stripped, during and after feeding. The temperament of the parents should be noted, as children of high strung, neurotic individuals are more prone to have this disease. The age is, usually, the second half of the first month of life; the nature of the feedings, in-

cluding quality and quantity, may be noted, especially when the vomiting first appeared, whether it is collective or projectile, and its relation to feeding time. Signs of gastric discomfort, gastric distension, and peristalsis, particularly the area over which the peristalsis is seen, should be noted. The stomach should be palpated when it is active, to determine its size, and when empty, and the abdomen relaxed, to feel for the pyloric tumor, as the tumor is best felt when the stomach is empty. Tarr states that palpation of a tumor is the most positive evidence in favor of a real stenosis.

Aspiration of a measured feeding after two or three hours will demonstrate obstruction. Wall advises feeding breast milk, or malted milk, because the large curds of cow's milk interfere with the aspiration. The objection to a stomach tube is that it frequently curls up in the cardiac end of the stomach, and nothing is aspirated; its use may have a bad effect on the baby. The character and size of the stools, the amount of weight lost, and whether diarrhea or constipation is present, should be noted. Duodenal bougies were formerly used to determine the patency of the pylorus.

#### DIFFERENTIAL DIAGNOSIS

Congenital stenosis should be differentiated from vomiting of a simple gastric or gastro-intestinal disturbance, intestinal colic, other forms of obstruction, and the various food injuries. So-called pyloro-spasm is differentiated from a real stenosis because the tumor is said to disappear on relaxation, and is not constant. A rare form of obstruction was reported by Downes. The case presented vomiting, loss of weight, and symptoms of high obstruction in the intestine, without any palpable tumor at the pylorus. At operation, a heavy peritoneal band passed from a loop of ileum across the hepatic flexure of the colon, and was adherent to the duodenum, causing complete intestinal obstruction. Another case had a small tumor from the muscularis projecting into, and filling the lumen of the pylorus; the tumor was discovered at autopsy.

Allen reported a case of a baby three months old, who had, instead of the usual tumor, a distinct plication at the pylorus, the duodenum being bent forward, and adherent to the stomach for a distance of almost one-quarter of an inch. The area was hyperemic, and there were cobweb-like adhesions, which he separated at operation.

Other conditions to be differentiated are ulcer of the duodenum, congenital atresia of the esophagus or pylorus, or of the intestine below the pylorus, and congenital bands pressing on the duodenum. Tuberculous peritonitis, with a normal pylorus, has been found at operation supposedly for pyloric stenosis. Downes states

that a freely movable right kidney may be mistaken for the pyloric tumor.

Dean Lewis of Chicago operated on two infants having all the symptoms of pyloric stenosis, except the tumor, but found nothing abnormal in the abdomen. I know of a similar case which was operated on by a competent surgeon of a neighboring city, and no tumor was found.

#### PRE-OPERATIVE PREPARATION

The value of pre-operative measures to combat dehydration, loss of heat, and subsequent shock is absolutely essential to successful surgery, and cannot be overestimated. Palmer believes that a delay of twenty-four hours should be made to prepare the infant to combat acidosis and dehydration. He gives alkaline gastric lavage, as well as alkaline enemas, 30 to 75 cc. every three hours, and 30 to 50 cc. normal salt solution subcutaneously every three hours. He continues the subcutaneous injections for twenty-four hours after operation. These injections, of course, should not be given at, or near, the site of operation.

The baby's arms and legs should be wrapped in non-absorbent, warmed cotton-wool, and this retained by loosely applied bandages. Hot water bags should be placed under and around the operating table. The temperature of the room should be between seventy-five and eighty degrees Fahrenheit.

The stomach should be emptied of food residue and gas by lavage with a stomach tube just before operation, if the infant is not extremely weak. If bismuth has been given and is in the stomach, it should always be removed by washing out with a tube.

Salt solution should be given by rectum or under the skin. Morgan objects to the latter because (1) there is a certain amount of exposure (which is of no importance if the room is properly heated); (2) a tumor on the back is formed, which may not be absorbed in time, and may interfere with the child's position on the operating table. In the writer's opinion this is a poor excuse, for towels, sheets, or blankets may be folded to conform with the infant's contour, provided excessive amounts have not been injected; (3) subcutaneous fluid not infrequently works around to the ventral surface of the body and encroaches on the site of operation. (If the fluid is sterile, no harm can be done anyhow.) Morgan strongly advises that its use be postponed until after the operation. Stone thinks that fluid before operation is most essential, if the baby is dehydrated. Porter gives 500 cc. of normal salt solution intraperitoneally. Objection has been made by Downes to giving too much subcutaneous fluid after operation because of the tendency to bleed, if 150 to 200 cc. are given. Rectal injections may cause vomiting.

In the writer's opinion, up to 25 cc. of salt solution may be given under each breast to advantage. At operation, sterile salt solution may be poured in the abdomen before closure.

Tyrrell and Reynolds institute systematic gastric lavage and subcutaneous injection of two per cent glucose in normal salt solution until the elasticity of the skin has returned, for at least two days, not to exceed four days. Operation should never be undertaken as an emergency in their opinion.

The alkali intake should be increased, and the bowels open before operation. Alkaline gastric lavage may be given. Prevent air swallowing during nursing if possible. A routine x-ray of the chest should be taken for evidence of thymus enlargement, followed by x-ray treatment if found.

#### ANESTHESIA

In the writer's opinion, one-half per cent novocain local anesthesia is the method of choice; it prevents shock, and disturbs the infant the least of any anesthetic. Both these factors determine the success or failure of surgery in the serious cases.

Gray and Reynolds believe that nitrous oxide gas, and oxygen will nearly eliminate operative fatalities. Tyrrell and Reynolds state that chloroform and ether, or ether alone, are unsatisfactory. They believe that local and spinal anesthesia expose these infants to dangerous complications. The writer knows of no dangerous complication to local anesthesia, unless it be faulty preparation of the local anesthesia solution, or partial evisceration, both of which are easily avoided. Palmer and others use ether. Bevan writes that under no circumstances should an operation for this disease be done under a general anesthetic.

#### OPERATION

Stone states that there is no operation in surgery, including that of the gall bladder, in which the location of the initial incision is of so great importance.

The Fredet-Rammstedt operation is performed by making a small three-fourths to one-inch right rectus incision in the right upper quadrant, through the middle of the right rectus muscle, one-half above and one-half below the border of the liver. Hook out the tumor, splitting the serous and muscular layers longitudinally in the bloodless line along the upper and slightly posterior surface of the pylorus, without opening the mucosa of the duodenum or stomach, thus minimizing bleeding and infection. The incision in the linea alba more readily reopens, according to Dean Lewis.

Gray and Tyrrell emphasize six rules essential for success: (1) gentleness, (2) speed, (3) minimum manipulation, (4) prevent evisceration.

(5) pyloric incision placed as near the convex upper surface as can be conveniently reached, (6) the tumor divided throughout its whole length, and bleeding points from the cut edges controlled by fine mattress sutures of catgut. Palmer stops bleeding by using tabs of cotton, very small, or gauze, dipped in boiling water and applied to the bleeding points, as he finds that catgut ligatures cut through the edematous, butter-like tissues.

The most important dangers are: (1) wounding the mucous membrane, with resulting peritonitis, (2) incomplete division of the constricting muscle, (3) hemorrhage, (4) shock.

Green and Sidbury, after the pyloric muscle is split, insert a stomach tube and inflate the stomach with air, to determine whether or not any perforation of the mucous membrane has occurred.

Strauss splits the inner part of each side of the pylorus after making the longitudinal incision, and enlarges it with the handle of the knife; the omentum is sutured over the wound to prevent hemorrhage or leaking, and to give new blood supply to the split muscle.

Cupler of Chicago modified the Rammstedt operation by separating the serosa from the muscle, allowing coaptation of the edges of the serosa without tension, and permitting the muscle to remain in the separated position.

Downes sutures muscle fibres from the rectus over any bleeding point. Harrison covers the incision in the tumor with an omental graft. Ransohoff of Cincinnati believes that the Rammstedt operation has complete simplicity, and absolute efficiency, and is one of the most perfect operations, and all its modifications are unnecessary.

Weller Van Hook thinks that plastic work on the pyloric muscle is not only unnecessary, but mischievous. In true stenosis, the muscle is too stiff and brittle for plastic work.

Palmer states that the technical error of opening the duodenum requires a plastic operation, or a gastrojejunostomy. Such is not the case, unless a very large opening is made. He recommends using No. 0 chromic catgut, with silk-worm gut figure-of-eight fascial sutures. Allen of Philadelphia uses through-and-through silk-worm gut sutures, removed in ten days or two weeks.

Very careful closure of the incision is essential. It must be sutured in layers, with extreme care, to prevent infolding of the peritoneum between the muscles, and resulting evisceration later.

Cupler closes the skin with linen, and reinforces the abdomen by means of a wide circular adhesive band. This is objectionable, as it may cause vomiting, as does a tight belly-band.

It is easy to roll the peritoneum in between

the muscles improperly, if through-and-through stitches are taken. Formerly the stomach was opened, and a sound passed through the pylorus after the muscle was divided, a procedure now obsolete.

#### POST-OPERATIVE CARE

Strauss gives one ounce of salt solution by rectum every three hours. When the child wakes, one drachm of mother's milk every two hours, and one drachm of water in the interval, increased so that in twelve hours the patient gets three drachms every two hours; in thirty-six to forty-eight hours, one ounce every two hours. Overfeeding should be discouraged, as edema may recur at the operative site, and cause a return of the symptoms. The results of a good operation are often offset by overfeeding. This is the most important point of the after care. Underfeeding can do no harm. Curtis begins with breast milk diluted one-half with water.

Morgan urges maintaining body heat, wrapping the infant in a warm blanket, or cotton jacket, and the bed well equipped with hot water bottles.

The position of the baby is important. For the first hour or two, the head of the bed should be lowered to prevent aspiration of mucus in the larynx. After nourishment is taken, raise the head of the bed, and keep the infant in a semi-erect position, which assists in emptying the stomach, and permits the escape of gas by mouth.

Stimulation with normal salt solution, or glucose solution, is always indicated,—120 to 240 cc. are most commonly used. Results from enteroclysis are so uncertain that its employment cannot be recommended according to Morgan. Epinephrin subcutaneously is the most satisfactory because of the rapidity of its action. Caffein and atropin and camphor in oil subcutaneously are sometimes valuable; by mouth dilute whiskey or brandy are of questionable value. The value of blood transfusion is very questionable as a stimulant, according to Morgan.

#### TEMPERATURE REACTION

Post-operative rise of temperature is to be expected in nearly all the cases, caused by (1) reaction from the shock of the operation; (2) subcutaneous salt solution and other stimulants; (3) artificial heat; (4) cotton wadding wrapped about extremities; (5) absorption of too much food passed down into the starved bowel. Convulsions may result from the same cause. The average temperature is 103; it may be 106°. There is no evidence that it appreciably influences the mortality.

Feeding, (Morgan). One hour after operation, 16 cc. water given when out of ether; one hour later, 12 cc. breast milk mixed with 4 cc.

water, using a medicine dropper; breast milk every three hours, alternated with water; twenty-four hours after operation, 16 to 24 cc. undiluted breast milk every three hours, and similar amount of water between feedings. After forty-eight hours, 20 to 30 cc., and at the end of seventy-two hours, 30 to 45 cc. at a feeding. Keep breast milk going with a vigorous baby, or use a breast pump. Baby to breast in one week after operation.

Post-operative vomiting may be due to: (1) gas in gastro-intestinal tract; (2) incomplete severance of muscle fibers; (3) too rapid increase of feeding; (4) peritonitis; (5) too tight belly band or adhesive strapping. No binder should be used.

A narrow strip of sterile gauze is held on the wound by adhesive strips. This gives early warning of bleeding, if there is any oozing from the skin, or of the wound reopening, a thing which wouldn't be noticed if a large dressing were applied. Stitch abscess and parting of the abdominal wound are to be watched for.

Diarrhea may result after operation, if the stomach is not washed out before operation. Porter believes that accumulated food in the stomach, contaminated with bacteria, on entering the intestine after the operative splitting of the stenosed pylorus, causes the diarrhea.

#### REPORT OF CASE

The following private patient is of interest because of the extreme degree of emaciation, the infant only weighing four pounds, a loss of fifty per cent of its birth weight which was eight pounds.

An early diagnosis of congenital pyloric stenosis had been made by Dr. Mitchell Sisson, who had delivered the mother and cared for the baby. The parents were young, temperamental Italians, having their first baby, a normal delivery. The baby was a breast-fed boy, and did well until the tenth day, when he began to vomit. Various kinds of feedings were tried, including breast milk from an aunt of the baby, all were of no avail, as the vomiting became explosive and progressively worse. Operation was refused until the twenty-first day, when it was very evident that the infant was rapidly failing, and the parents consented to an operation only when convinced that the infant was about to die.

Fluoroscopic and x-ray examination by Dr. Roy S. Perkins were unsatisfactory, as the infant vomited the barium meal as soon as it started nursing from the bottle.

Dr. Robert Curtis, in consultation, advised an immediate operation because of the very poor condition of the patient. The baby appeared like a wizened, dried-up old man, with sunken eyes and fontanelles. Its face was wrinkled and pale; its cheeks were drawn; it did not notice anything, and appeared listless. Marked lack of body fluid was evident; there was suppression

of urine, and the bowels had not moved for several days. There was a very evident olive-shaped tumor in the epigastrium, with visible gastric peristalsis.

Twenty-five cc. of salt solution was injected under each breast, and the infant's extremities were wrapped in warmed cotton and bandaged. Under one-half per cent novocain anesthesia, on a table heated with hot water bottles, at the Riverbank Private Hospital, a two-inch right rectus incision was made in the right upper quadrant of the abdomen. The rectus muscle was retracted outward, and the peritoneum opened. A few drops of ether were given at this point. The stomach was much distended, well below the umbilicus. The pyloric tumor was delivered through the wound, hard as a rock, smooth, white in color, and the size of a small olive. The tumor was split longitudinally with a sharp knife; gastric contents then escaped from the stomach into the duodenum. Inadvertently the lumen of the duodenum was opened, while enlarging the incision on the duodenal side, to make sure of division of all the tumor; a purse string of linen repaired this defect. There was no bleeding, so the pylorus was replaced in the belly.

The peritoneum and fascia were approximated with No. 2 iodized catgut, interrupted mattress sutures. The skin was sutured with interrupted catgut sutures of the same material. A small sterile gauze strip was applied with adhesive. The baby was returned to a clothes basket warmed by hot water bottles, in a specially heated room, and given drachm doses of breast milk and water, equal parts, every two hours. This dose was increased a drachm each feeding until two ounces were given at a time.

There was no post-operative vomiting, except on the third day, and this was due to too tight a belly-band, the removal of which eliminated the vomiting. The baby urinated on the dressing, subsequently there was some separation of the skin edges of the wound, with some redness and serous discharge, perhaps due to the novocain infiltration. Adhesive strapping approximated the skin edges satisfactorily.

The baby gained steadily, and was discharged against advice on the tenth day after operation.

#### END RESULT

When seen one year and six months later, it weighed thirty pounds and was in excellent condition, having no recurrence of symptoms.

#### CONCLUSIONS

1. An early diagnosis is most important; careful medical treatment should be supplemented by surgical intervention before complete obstruction occurs.
2. Intelligent pre-operative and post-operative care is very essential.



3. Novocain is the anesthetic of choice.
4. The Fredet-Rammstedt operation needs no elaboration or modifications, as advised by Strauss and Cupler.
5. A gastroenterostomy is not always necessary, even if the lumen of the duodenum is opened inadvertently.

#### BIBLIOGRAPHY

Allon, F. O., Jr.: *Ann. of Surg.*, 1919, lxxix, 531.  
Cupler, R. C.: *Surg., Gyn. and Obstet.*, 1918, xxviii, 223.  
Downes, W. A.: *Surg., Gyn. and Obstet.*, 1916, xxii, 251.  
Downes, W. A.: *Jour. A. M. A.*, 1920, lxxv, 228.  
Dunn, C. H., and Howell, W. W.: *Arch. of Pediatrics*, 1915, xxxii, 426.  
Gray, H. T., and Reynolds, F. N.: *Brit. Med. Jour.*, 1921, ii, 591.  
Green, T. M., and Sidbury, T. B.: *Surg., Gyn. and Obstet.*, 1919, xxviii, 159.  
Hass, S. V.: *Arch. of Pediatrics*, 1919, xxxvi, 516.  
Hagard, W. D.: *Jour. A. M. A.*, 1918, lxxi, 810.  
Hagard, W. D.: *Southern Med. Jour.*, 1918, xi, 506.  
Hill, R.: *Surg., Gyn. and Obstet.*, 1914, xviii, 616.

Holt, L. E.: *Jour. A. M. A.*, 1917, lxxvii, 1517.  
Keibel, F., and Moll, P. F.: *Manual of Human Embryology*. Phil. and London, 1912, ii, 353.  
Lewis, D., and Grulee, C. G.: *Jour. A. M. A.*, 1915, lxiv, 410.  
Morgan, E. A.: *Amer. Jour. Dis. of Children*, 1916, xi, 245.  
Morse, J. L., Murphy, F. T., and Wolbach, S. B.: *Boston Med. and Surg. Jour.*, 1908, clviii, 486.  
Palmer, D. W.: *Ann. of Surg.*, 1917, lxxvi, 428.  
Palmer, D. W.: *Arch. of Pediatrics*, 1922, xxxix, 582.  
Porter, L.: *Arch. of Pediatrics*, 1919, xxxvi, 585.  
Pritchett, J.: *Kentucky Med. Jour.*, 1921, xix, 731.  
Ranschoff, J. L., and Woolley, P. G.: *Jour. A. M. A.*, 1917, lxxvii, 1543.  
Sauer, L. W.: *Arch. of Pediatrics*, 1918, xxxv, 285.  
Scudder, C. L.: *Ann. of Surg.*, 1914, lix, 239.  
Scudder, C. L.: *Surg., Gyn. and Obstet.*, 1912, xiv, 373.  
Scudder, C. L., and Quinby, W. C.: *Jour. A. M. A.*, 1905, xlv, 1665.  
Strauch, A.: *Jour. A. M. A.*, 1915, lxv, 678.  
Strauss, A. A.: *Jour. A. M. A.*, 1915, lxv, 1532.  
Strauss, A. A.: *Jour. A. M. A.*, 1918, lxxi, 807.  
Tarr, E. M.: *Arch. of Pediatrics*, 1919, xxxvi, 154.  
Thomson, J.: *Brit. Med. Jour.*, 1921, ii, 589.  
Tumpeyer, I. H., and Bernstein, M. A.: *Amer. Jour. Dis. of Children*, 1922, xxiv, 306.  
Wall, J. S.: *Arch. of Pediatrics*, 1919, xxxvi, 193.  
Wollstein, M.: *Jour. Dis. of Children*, 1922, xxiii, 511.  
Young, H. H., Frontz, W. A., and Baldwin, J. C.: *Jour. of Urology*, 1919, iii, 289.

## TUBERCULOUS POLYSEOSITIS WITH REPORT OF A CASE

BY B. R. WHITCHER, A. B., M. D.

[From the Department of Pathology and Bacteriology, New York Post-Graduate Medical School and Hospital]

WHILE it is by no means an uncommon occurrence for a person, especially one past middle life, who has previously appeared to have been in good general health, to begin complaining of gradually increasing symptoms of discomfort about the heart, and occasional distress in breathing, with a slight amount of cough, but raising little or no sputum, these symptoms are associated, in the vast majority of cases, with gradual decompensation of some obscure valvular lesion with resulting chronic passive congestion of the lungs or with cardiac asthma or pericarditis with effusion and, in rather infrequent cases, these symptoms may, in reality, be due to tuberculous infection in which the serous membranes are extensively involved. Few or no indicative symptoms of actual tuberculosis may be present, and on account of the insidiousness of the onset and the amount of cough being so slight and the cardiac symptoms so pronounced, the likelihood of the condition being tuberculous is often overlooked, especially in the more chronic forms.

Under rest and the proper medicinal treatment an apparent improvement may take place, but later the symptoms of cardiac distress and fullness in the chest return and the patient begins to show signs of epigastric dullness and gradually increasing fullness in the abdomen and a little later ascites develops. The patient may show few or no pulmonary symptoms except gradually increasing distress in breathing from increasing pleural exudate or from pressure due to increase of pericardial exudate, and he may eventually die either from cardiac embarrassment or from complete intestinal atony, with general auto-toxemia. On performing the autopsy, it will be found that there is a large

amount of serous and fibrinous exudate with enormous thickening of the peritoneal, pericardial and pleural coats. The condition is then revealed as a very marked generalized tuberculous of the serous membranes or tuberculous polyserositis.

At the New York Post-Graduate Hospital there has recently been under observation a case of this progressive polyserositis in a man seventy-one years of age, with insidious onset, gradually increasing cardiac discomfort, and sensations of fullness in the chest, with marked ascites developing later, but practically no pulmonary symptoms except for a slowly developing shortness of breath from pressure due to cardiac enlargement and increase of pericardial exudate and only a very slight cough.

The patient, Joseph W. aged 71, first came to the hospital on July 2, 1923. He complained of shortness of breath which had been gradually developing during the previous six weeks, and which was most noticeable when he walked rapidly or went upstairs. This dyspnoea had become so bad at times that the patient had had to crawl upstairs on his hands and knees, but it had grown no worse during the last two or three weeks before entering the hospital. At the time the dyspnoea began, the patient's feet became painful with slight swelling of the ankles, the latter symptom lasting only a day, but the pains continued to occur when the patient walked. He also noticed that his appetite was not as good as formerly and that he had occasional pains in the epigastrium after eating, but no vomiting. He had no cough and no expectoration and his general health had been good, up to his present illness. The family history was negative, there being no history of tu-

bereulosis, carcinoma, or heart or kidney disease.

The physical examination showed a poorly nourished and emaciated elderly man. The antero-posterior diameter of the thorax was increased. The lungs showed hyperresonance throughout, with many crepitant râles in the left base and axillary line, and signs of fluid in the right base.

The area of cardiac dullness showed marked enlargement on both right and left sides, in all the interspaces, suggesting a large amount of pericardial effusion. The apex beat was invisible and could not be palpated and the apical sounds were indistinct, but those at the base and over the large vessels were fairly distinct, the aortic second sound being more distinct than the pulmonic. On X-ray examination the heart shadow appeared to be greatly increased in size, with broadening and knuckling of the aortic arch. The pulse was regular and of good volume and tension. The radial vessels showed beading.

The edge of the liver was palpable, extending nearly to the anterior superior iliac spine. The spleen was not palpable. Shifting dullness was present in the flanks. No masses or areas of tenderness were found on palpation. Examination of the gastro-intestinal tract by the X-ray showed the stomach small in size. Its mobility appeared fair, but that of the pylorus and of the bulb was somewhat limited. The intestinal peristalsis was sluggish and irregular in action with delay in evacuation. The examination suggested a mass in the right upper quadrant displacing the hollow viscera slightly downward and to the left. Whether this was an enlarged left lobe of the liver or a tumor mass of some sort could not be determined by the X-ray. The patient's condition at the time was diagnosed as pericardial effusion and right hydrothorax with congestion of lungs and liver, cirrhosis of liver, emphysema and arteriosclerosis.

On July 5 thoracic paracentesis yielded 350 cc of opalescent, straw-colored fluid. A complete blood count on July 10, showed 4,192,000 red cells per cu mm, 5,600 leucocytes, 65 percent hemoglobin, polynuclear neutrophils 86 percent, small lymphocytes 6 percent, large lymphocytes 7 percent and basophiles 1 percent.

The patient remained in the hospital for a month, being treated by rest in bed, with potassium iodide, gr. XV, elixir peptenzyme 1 dram, heroin and terpin hydrate elixir 1 dram and later, after the twelfth of the month, regulin 1 dram and dilute hydrochloric acid, 20 minims in water, each of these being given three times a day at 8 a. m., noon and 6 p. m. He was also given tincture of digitalis 30 minims each day. He showed a gradual general improvement but no changes in the heart conditions since admission. On August 2 he was discharged with a diagnosis of myocarditis and chronic cardiac de-

compensation, with improvement of symptoms under treatment.

He returned to the hospital on September 1, complaining of epigastric distress and discomfort. Since two weeks after his previous stay in the hospital he had noticed a gradual and steady increase in the size of the abdomen. At this time he did not complain of any shortness of breath, or cough or palpitation. After admission he had frequent watery stools, some of them being involuntary.

A blood count taken on September 4, showed 4,100,000 red cells per cu mm, 6,000 leucocytes, 9.9 grams of hemoglobin per 100 cc, or 61 percent, giving a color index of 0.74; polynuclear neutrophils 60 percent, small lymphocytes 23 percent, large lymphocytes 8 percent, transitionals 6 percent and eosinophiles 3 percent.

On September 10 an abdominal paracentesis was done and 3,300 cc of fluid were obtained, with a specific gravity of 1012. It was found to contain 6,900 white cells per cu mm, of which 20 percent were polynuclears and 80 percent were lymphocytes. Examination for mitotic figures and other evidences of malignancy was negative. The fluid was found by the Wassermann test to be anticomplementary.

X-ray examination showed the cardiac area considerably enlarged in its transverse diameter. The areus was slightly broadened and knuckled. The markings of the bronchi throughout the chest were more distinct than normal, with bronchiectatic thickening and dilatation.

He was given magnesium sulphate, one ounce, and tincture of digitalis, min. XXV, each morning and, after the fourth day, potassium iodide three times a day, and after the eighth, diuretin, gr. XXV, three times a day, each at 8 a. m., 12 noon and 6 p. m. Toward the last, colonic irrigations were given, the last ones yielding very poor returns and on September 14, at 11 a. m., the patient died.

At 3 p. m., an autopsy was performed. The body appeared emaciated and poorly nourished. The thoracic muscles were small and flabby and the ribs prominent. The abdomen was considerably distended.

On incision, the abdominal cavity contained about 2½ liters of opaque, brown, foul-smelling fluid. The intestines were fairly smooth, somewhat waxy-appearing and dark green on their free surfaces and bound together in a solid mass by firm adhesions, the small intestines and colon being adherent to each other. This mass occupied the upper left abdominal quadrant, only the sigmoid and rectum being separate and occupying their normal positions. The stomach and intestines were removed en masse. The coils of intestine could be separated from their adhesions only with very great difficulty.

The liver weighed 1335 grams, appeared somewhat shrunken in size and was adherent to the diaphragm by a larger area of its surface than normal. Its free surface was covered with a

thick yellowish green layer of thickened peritoneum. The spleen weighed 295 grams and was dark red in color with large yellowish-white scars on its outer surface. Both kidneys showed deep congestion with prominence of the stellate veins.

On opening the thorax the anterior medias-

yellowish-brown fluid. The parietal and visceral layers of the serous coat were yellowish white, waxy in appearance and roughened. The heart weighed 495 grams. On section the visceral layer of pericardium was found to be 3 to 7 mm in thickness. The walls of the coronary arteries were very thick and calcareous and the

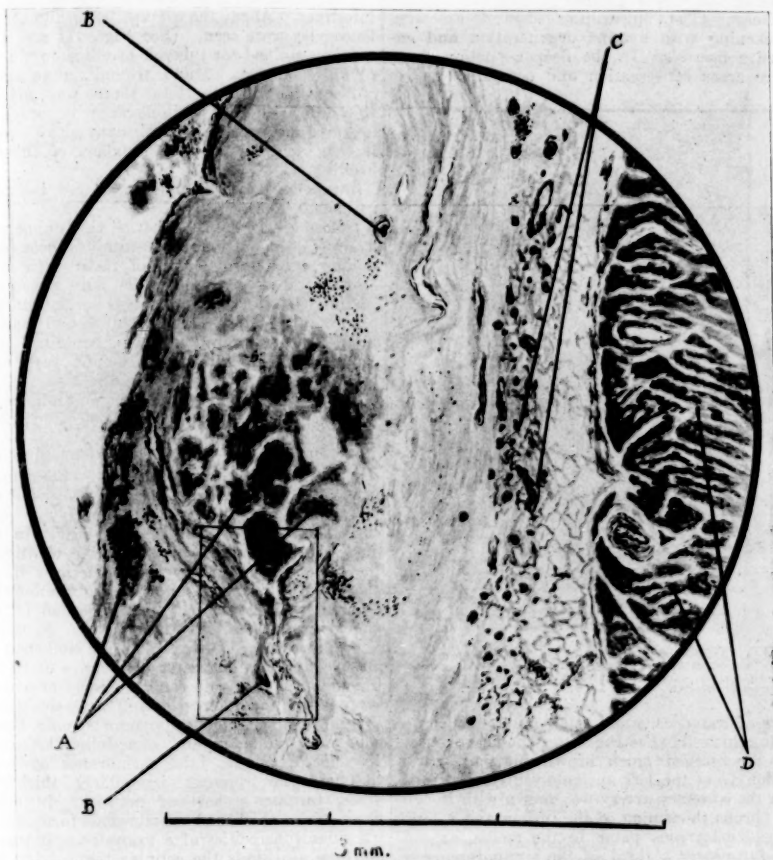


FIG. 1. Section of heart showing pericardial surface. A. Areas of caseation and necrosis. B. Langhans giant cells. C. Epipericardial areolar tissue with capillary congestion. D. Heart muscle fibre bundles.

tinal surface of the pericardium was adherent to the chest wall. The right pleural cavity contained about 400 cc of semi-transparent yellowish-brown fluid and the left one 100 cc of similar fluid. Both lungs were adherent to the pericardium by their mesial surfaces.

The parietal pericardium on section was 4 to 5 mm thick. The sac contained about 600 cc of

anterior flap of the mitral valve contained an atheromatous patch 18 x 14 mm. The aorta showed several atheromatous patches and the walls of the innominate, left common carotid and left subclavian arteries were very thick and inelastic.

The lungs were separated from their pericardial adhesions with difficulty. The left lung on

section showed many small hard nodules in the upper lobe and many small rounded fibrosed areas in the lower lobe.

The histological findings were as follows:

**Pericardial sac:**—The parietal pericardium was very much thickened and necrotic and contained many small tuberculous foci with giant cells in the deeper portion.

**Heart:**—The epicardium showed excessive thickening with hyaline degeneration and extensive necrosis. In the deeper portion there were areas of caseation and occasional collec-

infltrated with round cells and polynuclear leucocytes.

**Small Intestine:**—The subserous coat was thickened, with an external zone of necrosis. Beneath this necrotic zone were numerous small caseating tubercles blending with each other, especially where one loop of intestine was adherent to the other, and practically encircling the intestine. About these areas large numbers of leucocytes were seen. (See Figs. III and IV). Sections stained for tubercle bacilli showed large numbers of them in and around the tubercles.

**Large Intestine:**—Here there was marked thickening of the peritoneum with areas of necrosis and necrobiosis and many small caseating tubercles, with large numbers of tubercle bacilli.

**Liver:**—Contained a few scattered tubercles in its substance.

**Spleen:**—The capsule showed thickening with hyaline degeneration and contained occasional miliary tubercles. A few of these latter were also scattered throughout the pulp substance.

**Kidneys:**—Marked vascular engorgement was present throughout the medulla and cortex. Rarely a uriniferous tubule contained calcareous deposit. No tuberculous foci were found in either kidney.

With these gross and microscopic findings the case was diagnosed as one of tuberculous polyserositis or polyorrhnenitis.

P. M. Feitu, in the Paris theses of 1820<sup>1</sup> described peritonitis as susceptible of a great number of complications, such as phlegmasias of the other serous membranes, as arachnitis and pericarditis, but most commonly pleurisy. In the discussion of chronic peritonitis he mentioned the latent type as appearing sometimes in an obscure manner with mild, but continuous abdominal pains, tumefaction, fluctuation of the abdomen and sensibility on pressure, especially lateral pressure. On opening the abdomen at autopsy there is a greater abundance of serous fluid than in the acute conditions and of a most disagreeable odor, less limpid and sometimes sanguineous. The peritoneum is more thickened, less reddened and sometimes blackened. In some cases the false membranes covering the intestines present irregularly thickened areas, forming a sort of network. In other cases these thickened membranes form small irregular tuberousities of a granular appearance and one also finds the cellular tissue lying behind or between the peritoneal folds as thickened and lardaceous.

Pierre Charles Alexandre Louis, in 1825, in his work on phthisis<sup>2</sup> stated that on following the different chronic affections of phthisis he had observed several cases of the outpouring of serous exudate into the abdominal cavity, varying in amount from one to eight pints. Frequently with this exudation of serum, a soft yellowish false membrane was found, or a certain quantity of thick odorless pus similar to that



FIG. 2. Area in pericardium, outlined in Fig. 1, seen under higher magnification, showing hyaline degeneration, caseation and necrosis. A. Langhans giant cell. B. Group of round cells. C. Necrotic material.

tions of round cells and small miliary tubercles with giant cells (See Fig. 1). The heart muscle fibres contained much brown pigment. In a section from the left auriculo-ventricular junction the coronary artery was shown with irregular fibrous thickening of the intima and a large ragged calcareous patch in the media, extending for nearly a third of the circumference of the vessel. Staining for amyloid degeneration was negative.

**Lungs:**—The pleura was slightly oedematous with diffuse round-cell infiltration and anthracosis. Rarely a calcareous nodule was seen. Scattered throughout the lung substance were many tubercles in various stages of development, some showing caseation and others calcification. Many showed anthracosis.

**Stomach:**—The peritoneal surface showed necrobiosis and the subserous coat was thickly



found in a warm abscess. In cases of diseases of the heart in which this serous exudate often occurs Louis had encountered this condition sixteen times in seventy-seven subjects, in almost the same proportion as in phthisical patients, but none of the former showed signs of tuberculous peritonitis, either in the way of semi-transparent granulations on the peritoneal surface or a thickened, false membrane applied to

tuberculosis as certain and easily explainable.

Bait<sup>4</sup> in describing tuberculous peritonitis stated that in some cases ascites is for a long time the only manifestation, developing slowly. Examination of the heart and of the urine will avoid confusion with dropsy subsequent to diseases of the heart and with that of Bright's disease. Its greatest frequency is from 32 to 38 years of age, rarely appearing in old age. It

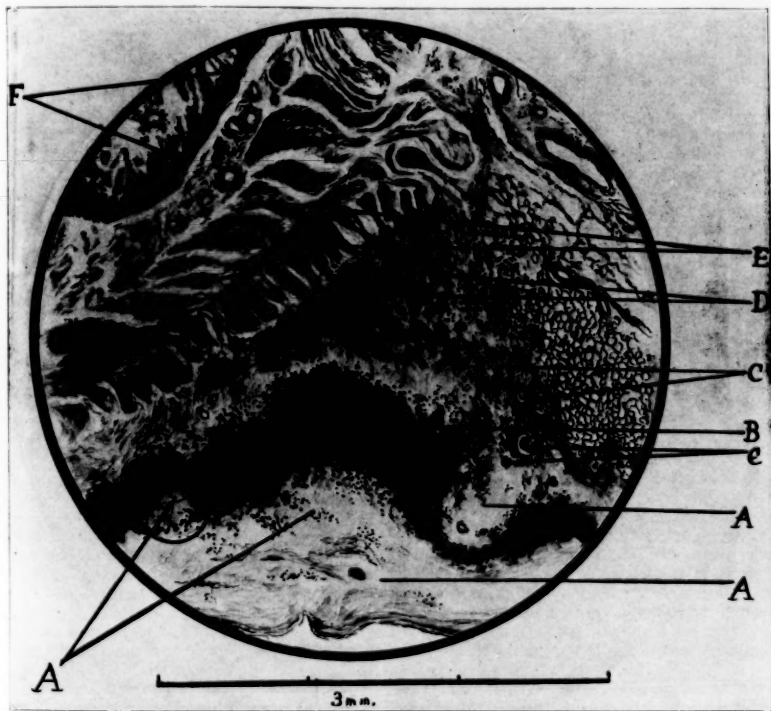


FIG. 3. Section of small intestine. A. A. A. Areas of caseation in peritoneum. B. Zone of lymphocytes and endothelial cells, many of them disintegrated. Here many tubercle bacilli were found. C. Langhans giant cells. D. Infiltration of tissue with lymphocytes and occasional polymuclear leucocytes. E. Muscular coats. F. Mucosa.

that surface. Therefore, he regarded these latter types of exudate as occurring exclusively in phthisical patients.

Mouroux<sup>3</sup> in 1883 reported several cases of cirrhosis of the liver in alcoholics which were found at autopsy to be accompanied by tuberculous peritonitis and he showed that when a patient developed hepatic cirrhosis, the condition brought about an irritation of the peritoneum which lessened its resistance and made it an easy prey to tuberculous infection and therefore he regarded the influence of cirrhosis of the liver upon the development of peritoneal

occurs twice as frequently in men as in women. The existence of pleural effusion, either before or after the appearance of the peritonitis, is extremely frequent and a great aid to diagnosis, and tuberculous pericarditis, though much rarer, is of the same value. This ascitic form of tuberculous peritonitis may complicate cirrhosis of the liver and obscure the diagnosis.

H. Curschmann<sup>5</sup> in 1884 described a case of a woman 56½ years of age who had had attacks of peritonitis beginning with rigor and pain in the abdomen and tenderness and swelling over the liver. Repeated paracenteses had

been done and there had been partial recovery. After a course of six and a half years the patient was taken with rigors, vomiting, remittent fever and an apparently fresh attack of peritonitis. The liver was small and hard but there was no cirrhosis in its substance. Curschmann described the condition as "Zuckergussleber." The pericardium was

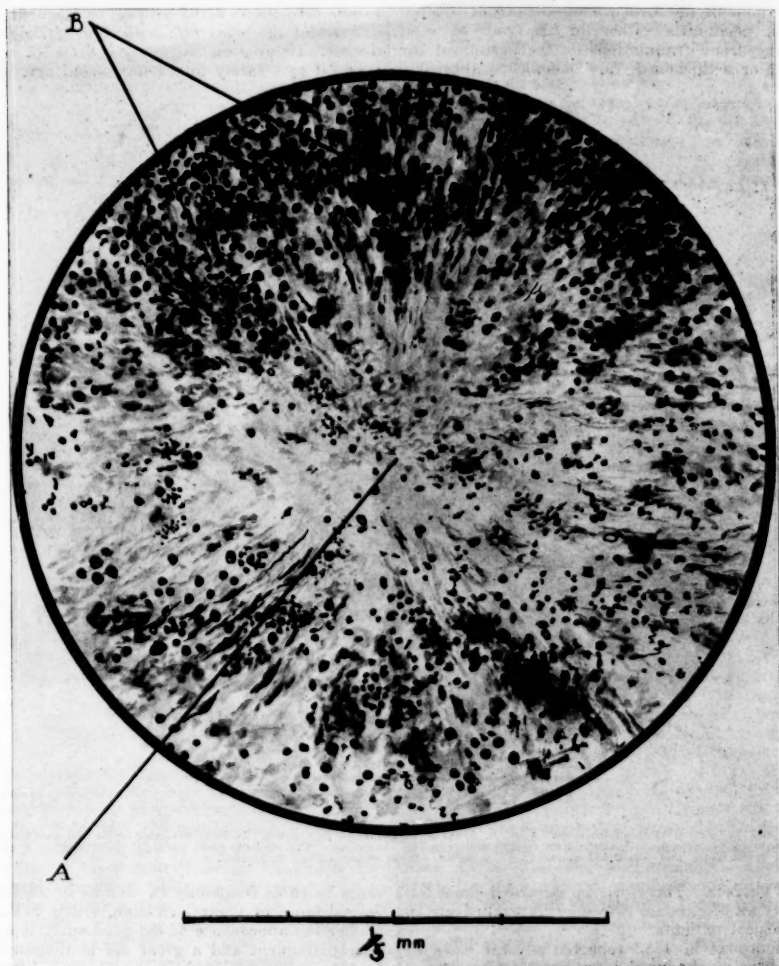


FIG. 4. Tuberculous caseating area from small intestine under higher magnification (outlined in Fig. 3). A. Area of necrosis. B. Zone of round cells and occasional polynuclear leucocytes.

tonitis, accompanied by pleurisy on the left side, and died. The autopsy showed the upper portion of the peritoneum to be thickened and of a bluish-white pearly appearance. This enveloped the anterior abdominal wall, the liver and the obliterated and the right pleura and tissues of the anterior mediastinum were thickened. The cause of death was pronounced a recent tuberculous peritonitis. F. A. C. Scrimger<sup>6</sup> describes a very extraordinary condition of the perito-

neum occasionally found, known as hyalo-serositis, polyserositis of "Zuckerguss" liver, and in these cases the pleura and pericardium, as well as the peritoneum, are often involved. This condition is characterized pathologically by a dense white coating, usually most marked over the liver and spleen, looking very much like the frosting on a cake and composed of dense hyaline tissue. According to Scrimger this thick hyaline envelope, frequently associated with chronic changes in the liver, causes obstruction of the portal system and the collection of fluid in the peritoneal cavity results. It is rarely distinguished from ascites due to chronic cirrhosis of the liver or chronic failure in the compensation of the heart.

F. Taylor<sup>7</sup> of Guy's Hospital, in 1900, stated that the frequent association of the serous cavities in a common lesion had long attracted attention, not only in combined tuberculous peritonitis and pleurisy, but in acute rheumatism, which often invades both pleurae and pericardium simultaneously, and pyemia which may attack everyone of the great serous sacs at the same time. But after all, according to Taylor, the tubercle bacillus is by far the most frequent cause of the disease. There have been cases of acute peritonitis in young people, apparently caused by appendicitis or intestinal obstruction, which have proven, on operation or after death, to have been tuberculous in origin. The original starting point of the inflammation may have been on the upper or under surface of the diaphragm, but on whichever side the infection starts, whether from a pleuritis accompanying pulmonary tuberculosis or from a diffuse peritonitis, it passes through the diaphragm by way of the blood vessels and is consequently apt to be distributed widely through the serous cavities as well as through the whole body.

A. G. Nichols<sup>8</sup> describes two cases of multiple progressive hyaloseritis in which he noted the formation of a dense hyaline membrane opalescent in appearance, somewhat resembling semisolid celloidin. In these cases the liver, spleen, lungs and heart were involved. On account of the thickened hyaline membrane the liver is called "Zuckerguss" liver, and where this organ is affected, the patient shows general weakness, dyspnoea and increasing and obstinate ascites. The process of membrane formation affects by preference the upper part of the peritoneum, the bases of the pleural sacs and the pericardium. In both of these cases sections were stained for *Bacillus tuberculosis*, but none were found. Nichols claims, however, that the membrane formation is due to microorganisms causing a low degree of infection.

A. O. J. Kelley<sup>9</sup> in 1903 discusses several cases of multiple serositis characterized clinically by marked ascites with little or no oedema of the legs, in which diagnosis of cirrhosis of the liver had usually been made and which, at autopsy, showed chronic obliterative pericardi-

tis with pleuritis, peritonitis and peri-hepatitis. From his observations it is evident that the clinical manifestations may vary, depending upon the serous membrane first affected, as, where the disease first begins in the peritoneum, ascites is the prominent symptom. On the other hand it must be granted that multiple serositis may begin in one or the other pleural sac.

Anatomically the distinguishing feature of these cases was a chronic fibrous inflammation of the several serous membranes of the body and there was a marked tendency for all of the serous membranes to become involved. Ascites was the striking clinical feature in this disease. In consequence of the inflammation, the peritoneum, in such cases, becomes a place of lessened resistance and for that reason, in many chronic infections, such as tuberculosis, the efforts of the peritoneum to remove the infective agent are unsuccessful or only partly successful. As a result, the region about the liver and the under surface of the diaphragm succumbs and a perihepatitis occurs and subsequently, through the peculiar lymph supply on the surface of the liver and under surface of the diaphragm, the infective agent may travel through the diaphragm and infect the pericardium or pleura, or both. Many of these cases are tuberculous in nature, resulting generally from extension of the disease from tuberculous mediastinal or peribronchial lymph nodes, and thus either an ascending or descending infection of the several serous membranes may occur.

Tuberculous peritonitis is most common between the ages of twenty and forty. The infection may reach the peritoneum through the blood vessels or else it may be found secondary to a tuberculous focus elsewhere in the body, as in the lungs, genito-urinary organs or intestines. It may occur in acute general miliary tuberculosis, or it may be a primary process. It may reach the peritoneum by continuity from the pleura or pericardium through the lymphatics of the diaphragm, or it may spread through the contained abdominal organs.

When occurring in acute general miliary tuberculosis, the parietal and visceral surfaces of the peritoneum are mottled with small gray tubercles with little local inflammatory reaction but in the most frequent forms of tuberculous peritonitis the viscera are bound together by numerous peritoneal adhesions which are often so firm that it is impossible to separate them. In these red and grayish-red adhesions, small nodules and caseating masses are found. In a small number of cases a fluid exudate is absent (tuberculosis sicca) but as a rule the spaces between the adhesions are filled with serous, serofibrinous, fibrino-hemorrhagic, fibrino-purulent or purulent exudate. Frequently the organs are so covered with a thick yellowish-gray fibrino-purulent mass that proper orientation is almost impossible. The omentum may be involved and become converted into a hard, thick

mass in the upper part of the abdominal cavity and the mesentery may become very much shortened. A generalized form of tuberculous peritonitis may be associated with the terminal stages of a chronic disease. This is more particularly the case in cirrhosis of the liver or chronic ascites of any kind, because of the lowered resistance following prolonged irritation of the peritoneum from the chronic inflammatory processes.

Ascites is usually present in these cases, with turbid yellow or blood-stained fluid, and the whole peritoneum becomes studded with countless grayish-white or yellow nodules lying close to each other, being most numerous near the diaphragm.

But, as has been said in the introduction to this article, a very marked case of tuberculous polyserositis may develop, with few or no objective symptoms of actual tuberculosis being present. The disclosure of the actual condition of tuberculosis often comes as a surprise, tuberculosis not having been suspected, because there had been no cough and no expectoration and the dyspnoea had been apparently due to the pronounced cardiac symptoms, but the history and post mortem findings of the patient show what extensive damage may be done to the

serous membranes by tuberculosis and the gradual and insidious progress of the infection.

The most striking features of the present case may be summarized as follows:—(a) The old age of the patient, 71 years, tuberculous peritonitis and polyserositis being very rare in old age. (b) The marked exudative type of tuberculosis with the extensive caseation and necrosis. (c) The large amount of fluid in the abdominal cavity and pericardial sac, and the lesser amount in the pleural sacs.

#### REFERENCES

- 1 P. M. Feltz: Dissertation sur la peritonite, considérée dans son état aigu et chronique. Paris Thèses, 1820, Vol. II, No. 49.
- 2 Pierre Charles Alexandre Louis: Recherches anatomico-pathologiques sur la phthisis, p. 151. Paris, Gabon et Cie, 1825.
- 3 Mouroux: Des rapports de la cirrhose du foie, avec la peritonite tuberculeuse. Paris Thèses, 1853, No. 417.
- 4 Victor Biat: Peritonite tuberculeuse a forme ascetique. Paris Thèses, 1854, V. 19.
- 5 H. Curschmann: Zur Differential Diagnostik der mit Ascites verbundenen Erkrankungen der Leber und Pfortaderstämme. Deutsche Medizinische Wochenschrift, 1884, Vol. X, p. 564.
- 6 F. A. C. Scrimger: Inflammations of the Peritoneum. Nelson's Loose-Leaf Living Medicine, Vol. V, p. 594.
- 7 F. Taylor: Polycystic degeneration or Combined Serous Inflammations. British Medical Journal, Part II, 1900, p. 1625.
- 8 A. G. Nichols: On a somewhat rare form of chronic inflammation of the serous membranes (Multiple progressive hyalinosclerosis). Studies from the Royal Victoria Hospital, Montreal, 1902, I, No. 3.
- 9 A. O. J. Kelley: On Multiple serositis—The association of chronic obliterative pericarditis with ascites. American Journal of the Medical Sciences, 1903, CXXV, p. 116.

## THE GENERAL PRACTITIONER AND INDIVIDUAL PREVENTIVE MEDICINE\*

BY J. PENTEADO BILL, M. D., DR. P. H.

*The Field.* Most physicians are prone to classify humans as either sick or well. The terms are only relative, to be sure, but convey a somewhat definite idea of health. We are agreed I think that people who make up our clientele are for the most part frankly needful of our services; that is, the disabilities have reached such a pass that they interfere with normal activities. I think we are agreed again, that those who come to see us are but a small fraction of the large number of ailing people. The latter include many who know they are under par, but who feel diffident about getting medical attention, whatever their reasons may be.

It is this great middle class of sufferers from disfunction, or dis-ease, laying between the sick and well, that I have in mind, and for whom I believe Individual Preventive Medicine to be indicated. In the past, this field for professional endeavor has been practically ignored. Where disfunction exists, restoration is surprisingly easy and satisfactory through Nature's powerful assistance. It seems to me therefore that we have at least two good reasons for giving this field our earnest attention.

Assuming our agreement on the above pre-

misses, let us see what can be done to restore the disfunction to normal.

*Individual Preventive Medicine.* Of Preventive Medicine as such you and I have a definite idea. We think at the same time of the public health. In it attention is directed toward the control of communicable disease primarily. By it the individual profits only indirectly. Our public health departments have been so active in the past generation that the laity has come to expect to be kept well.

If we undertake however to restore an ailing patient to well-being, our first reaction is one of uncertainty as to what to do, for our training has all been along the lines of pathology and symptomatic treatment. It is obvious that public health measures do not apply. The fundamentals of preventive medicine as applied to the individual are entirely different from those of public health activities, as they are from those of curative medicine.

They include a consideration of posture, diet, exercise, and daily habits, the abuse of which four factors is largely responsible for dis-ease or disfunctional conditions.

All of us are familiar with a type of patient whose ills find symptomatic expression in ways that do not permit of exact diagnoses. Luckily

\*Read before the Plymouth District Medical Society, Hanson, April 17, 1924.



the morning after effect of pre-Volstead days has in part disappeared. There are however many individuals in whom every morning has its after effect—who do not feel normal until they have been awake and active for some time. In such cases as well as in other types of long standing disfunction there may be only one ambiguous symptom or sign, such as lack of energy, indigestion, headaches, irritability, etc. The profession by and large does not do anything for these individuals. The layman knows it. The various cults will glibly explain the supposed reasons, but, more important still, will give the appearance of doing something for the supposedly underlying cause. The physician may prescribe laxatives, for example, but these are symptomatic in effect. The cause still remains, so far as his services have gone.

The profession can do more lasting good in these cases of below par-ness by so-called hygienic means than by any other method or therapy alone.

The inquiry must be directed along two different lines; first, a thorough physical examination, and second, a careful scrutiny of the patient's daily life. One is useless without the other. Frequently both must be further amplified by special examination of the nose and throat, pelvis, digestive tract, etc. Headaches, e. g., may be due to many causes. Perfunctory questions about the eyes, menses, bowels, diet, overwork, etc., as possible causes may not lead to a correct understanding of the problems. One must elicit all the facts, and form one's conclusions. Then, and then only, would an examiner be justified in reaching any decision as to the reason for the client's unease.

*Physical examination.* In my own work, the physical examination sheet is divided into sections, such as Appearance, Heart, Lungs, Mouth, etc. These in turn are divided into subheads, each qualified by printed adjectives in lighter type. According to the findings the appropriate adjective is under-scored while conducting the examination. Black ink is used for findings to be regarded simply as matters of record, while red is used for points which demand further scrutiny or correction. Space is reserved on this sheet for recording the necessary laboratory determinations. The adjectives used refer largely to locational or postural abnormalities rather than to strictly pathological findings for again I must emphasize the fact that most of this work is on patients who are still ambulant and for the most part engaged in their regular vocations; their conditions have not yet had time to become pathological to an appreciable degree. This type of blank is a great time and concentration saver. By it, one's attention may be continually directed toward the examining of the patient and not be diverted by mental search for descriptive English in which to record it.

It will at once be observed that possible findings cover many fields of special endeavor.

Postural defects may need orthopedic treatment, hernias surgical, pelvic the attention of the gynecologist, teeth the dental surgeon. In such event the patient sees a specialist in the given field with the understanding that a definite diagnosis may be made and treatment given if the condition warrants it. In this way the patient may be said to derive the benefit of group treatment, save that it is strictly limited to conditions needing such. There is no apparent need for having each client go from one specialist to another to be given his own type of examination. This saves reduplication of work as well as many repeated negative findings, obviously a time and money saver all around. Further, the proportion of such special examinations is rather small.

The physical examination may be likened to the intended purchase of a used car. Everything that does not appear to be in proper functioning condition may be looked over specially, as e. g., the ignition by an ignition expert. But no amount of looking under the hood or functional testing is going to inform the purchaser how the car operates on the road. The same is true with the used human engine. There are many who have driven their own human machines in a faulty way, finally realizing vaguely that something is the matter with themselves. They usually want something done about the situation. Have we any right to reassure them and let them go, as we have been prone to do?

*Hygienic examination.* Here is where the second line of inquiry—the examination by the hygienist—demonstrates its value. The physical may show practically nothing worthy of note, while the investigation of the individual's daily life may show glaring faults. I do not intend to imply that the corrections will bring about well being in all instances, but it will result in such profound changes in the client's feelings as to be striking.

Right here is where I believe most physicians fall down. I myself tried verbal inquiry into the patient's daily life, but found that the procedure gave me no clearly defined picture, besides taking up too much time. I give the patient instead a printed blank with some fifty questions at the time of his physical, and arrange for its return with urine and stool samples either before or upon his second visit for a discussion of his case. People like to go into detail about themselves. I'd much rather they would do it on their own time than on mine. When the blank is returned, a rapid resumé with the red pencil enables one to check off the points requiring correction. These, together with those on the physical sheet, in connection with the laboratory and other findings enable one to lay out a reconstructive regime whose effect is usually gratifying to all concerned.

There are one or two pertinent observations that might be made as a result of going over

many people in the indicated below par condition. The most outstanding feature is viscerop-tosis. In the erect posture, lower stomach borders and transverse colons well within the pelvic brim are not at all uncommon. It is not surprising then that so many instances of digestive tract disfunction are to be met. This condition can to a great extent be remedied mechanically, first through proper posture and postural exercises, second through corsets and belts, and third, through changing the character of the diet. It is almost a foregone conclusion that below par-ness is associated with viscerop-tosis, and an X-ray examination of the digestive tract, prone and standing, is now given as a regular part of our investigation. This is done by my associate, Dr. Herman A. Osgood.

Viscera displaced by the effect of gravity are slow in functioning. While a barium meal will show stasis, a useful and simple test is to have the patient chew and swallow a half ounce of charcoal, while following his accustomed habits, and to make note of the time till the stool loses its black color. This time factor in my records varies from six hours in a case of nervous diarrhea to over four days in a case of obstinate constipation, in spite of daily movements.

Another matter for comment is fecal occult blood. The persistence of positive tests in patients with vague gastric symptoms who have been on a meat free diet for several days has led to its adoption as a routine. In many such cases the X-ray will give no evidence of ulcer, but one is forced to the assumption of a pre-ulcerous condition through the relief brought about by an ulcer regime and resulting disappearance of occult blood.

Many have asked me how to get into this line of work. Some cases are of course referred; most such in the average doctor's practise he can himself direct into this channel. The following cases will show how it can be done.

Case 1. A. R. H. act. 41. Travelling salesman. One night I was called out in the town where I live to see this patient who had gone to the toilet and had fainted. He was conscious when I arrived. His picture was remarkable in no way save for a slight pallor, chilliness, and a somewhat feeble pulse. This was the fifth of similar attacks in the previous three years. He was given a stimulant and put back to bed. I then told him there were two possible things to be done for him, one was to treat him for his present difficulty, due to careless bowel habits while on a trip from which he had just returned, and the other was to go over him from crown to toe, and from one morning till the next morning, with a view of ascertaining and removing the cause of these attacks and his feeling of apprehension regarding them. He chose the latter alternative.

He was also told that this procedure would

require special methods of inquiry, different from those to which he was accustomed in his previous dealings with physicians, that it would take considerable of his time, as well as mine, and that he must expect to pay accordingly. The point was also made that whatever he so paid would be best regarded in the light of health insurance, for there was a reasonable certainty that the program to be outlined to him would eliminate once and for all any recurrence of his temporary disability, if he would follow it intelligently and consistently. He was told in addition that he had been years in getting into such a pass, and that it would be months before he could himself feel betterment of his body functions, and that in the interim most of the onus of responsibility would be on his own shoulders.

He was further told that the regime would not hinder but would amplify the services of his family physician whom he must consult for acute conditions requiring treatment, and who would be informed of the regime as outlined. Parenthetically in referred cases the report and recommendations are sent directly to the physician. The preceding paragraph embodies features in the conversation, which introduces this line of endeavor to the patient perhaps for the first time, which I regard as absolutely essential to a clear understanding of what is to be done and what the patient may expect of it.

If the physician attempting this specialty does not himself feel the dignity of his efforts, the patient will not respond accordingly. The original investigation takes time and study. The doctor should be remunerated accordingly for the work can not be done on the office call basis.

The outstanding features of the examination of this case, that is, those which in my opinion warranted the red pencil, are as follows: sallow skin, drooping posture sitting or standing, round shoulders, a single inch difference between chest and abdominal girth, and apprehension. Surely not very much in the way of physical findings. The hygiene blank had more to question. As his wife said, a meal wasn't a meal without meat and potatoes. His diet was varied and generous. His food was preferably highly seasoned and heavily salted. He frequently experienced acid eructations after meals together with headaches. He used tea. His baths were weekly events with warm water, never with cold. He had no hobby and never did real muscular work. His posterior nares were continually scratchy and catarrhal in winter. His home is heated with a hot air furnace. Despite the heavy underwear he used in winter he suffered from the cold.

The charcoal test first showed in 31 hours after ingestion and disappeared after 55 hours, with daily movements. The laboratory findings were not remarkable.

Instruction directed to the correction of his below-par-ness included a consideration of diet,

bowels, baths, liquids, avocation, posture, and exercise. Diet. He was instructed to have for breakfast fruit, cereal with cream and sugar, toast with butter, eggs poached or boiled, and a cup of coffee with cream and sugar. His dinner at night was to consist of his wonted foods save meat which was restricted to one meal a week, while he was told to favor salads as much as possible for his luncheon, using lime juice and olive oil for dressing. Altogether the amount of food prescribed for him was considerably less and of a more easily digestible character than that to which he had been accustomed. He was told to eliminate tea entirely from his dietary, but to continue his habit of 2 to 3 glasses of water with his meals. His bowel habits were irregular in spite of his daily movement, as shown by the charcoal test. They were eventually regulated in the following way. He was instructed to regard a movement after each meal as the ideal toward which to strive, and to go to toilet after breakfast and dinner, whether the desire be present or not, with the idea of accustoming his digestive tract to emptying itself at these times. As an aid to more prompt evacuation, powdered agar was to be used every morning, but gradually to be discontinued.

He was told to take a daily warm sponge and to follow it with a cool sponge and vigorous rub-down to tone up his vaso-motor system.

For an avocation he was asked to find an outdoor activity which would give him healthful exercise an hour or more daily if possible.

In regard to posture, the mechanism and relationship between visceroptosis on the one hand, to proper posture on the other, with its effect on the abdominal wall was explained to him. Postural exercises were outlined. He was told to keep his gaze on a level with his eyes, to keep his shoulders back, his belly wall in, and to let the use of his pencil, which he employed frequently during the day, suggest proper posture to him. He was told to come in at intervals of a month or longer, to report progress. I saw him a few days ago, after two years of this supervised regime of functional reconstruction, and his own words were "I never imagined it would be such a cureall." He has very regularly two movements a day, sometimes three. He has lost his craving for meat, eats less food, is hungrier at meal time, and as he says, "am on my toes all the time." His morning loginess, dizziness, headaches, indefinite pains in his joints and all the rest of his symptoms have disappeared. To be sure there is a swift return of them if he lapses into his old manner of living, but he now knows what to do for himself.

Case 2. J. C. M., aet. 47, plumber. This man came because he knew he would be given a "health examination." He had not worked during the winter for seven years because, as he termed it, he suffered with rheumatism. His complaints were "headache, rheumatism, diges-

tion and condition of his feet." His physical examination showed round shoulders, drooping posture sitting or standing, toes out, moderate pes planus, hemorrhoids, pyorrhea. He was apprehensive and needed glasses. His tabulated daily life showed errors in diet, bowel habits, baths, liquids, avocation, posture, exercise, and hygiene of occupation. He had been overfeeding and undereliminating.

Summarized in more detail, he had used three hearty meals a day, two of them with meat, bolted his food, overemphasized white flour products, and was very sparing with fruit and green vegetables. His total liquid intake was three cups of strong tea and four glasses water. As to particular foods hurting him, he said that pork and fried potatoes gave him gastric distress and headaches. It was 1 p. m. daily before he really felt normal. His baths were limited to a Saturday night affair. His reactions to temperature, minor respiratory infections, etc., all showed the lowered vaso-motor tone and mucous membrane resistance.

The essentials of his recommended regime are these: Diet; two meals a day with a light warm lunch at noon only as felt to be absolutely necessary. This matter was of course entered into in detail as to character and amounts of food. Liquids; coffee only at breakfast, no tea, minimum of five glasses water with and between meals, and warm beverage at lunch and dinner. Bathing, warm and cool sponge followed by dry rub daily. Bowel habits; toilet after breakfast and dinner as daily habit procedure till twice a day habit established, which is usually fairly easy on a change of diet with mild therapy. His usual posture in connection with his work was on his knees and toes. The effect of this posture on visceroptosis and his feet was explained to him and exercises outlined to him to straighten out his back, assist in relieving the ptosis, and permit him to walk with his toes flat on the ground, which he could not do. Eighteen months later he said he had worked all winter without missing a day. He said he looked and felt twenty years younger, and his symptoms have disappeared. That is his story today, three years after being examined.

Case 3. M. C. M., stenographer, aet. 27. Her story was one of overweight, occasional headaches, and irregular menses, and is one very commonly encountered in any doctor's practise. The findings on examination were overweight, drooping posture, beginning pes planus, T. 100.4, ammoniacal breath, pyorrhea. Her questionnaire showed sluggish menses with prolonged function and intervals. Bowels acted daily, but the charcoal test showed 20 to 36 hours necessary for complete elimination. Muscular activity was only that necessary in her office work.

Recommendations were made in regard to posture, postural exercises, corseting, manner of walking and breathing, and bathing. Because her diet and elimination were obviously at fault,

she was advised in detail in those respects. The teeth were to be overhauled by a competent dental surgeon. It was also pointed out that betterment of body functions would undoubtedly make for a change for the better in the menstrual history.

Sixteen months later, she had lost 28 pounds, blood pressure was lowered 15 mm. to 135 systolic, pulse from 116 after she had finished with her examination to 72, headaches had disappeared, while the menses were regularly about 29 days apart with little discomfort.

The above three cases are fairly simple in character. The changes wrought for the better were done with hygienic means alone, save for dental work. These people all returned at intervals varying from one to several months to report on their condition. This is necessary for what improvement does occur is gradual, and sometimes needs to be hastened by changes in the daily regime. One of the most difficult things to make a patient understand in this work is that it is going to take time before they themselves will admit any definite change for the better. As some one has put it, "tissues have habits and memories," and a faulty function has considerable inertia. Patients can be told to advantage that if they have been years in getting into their present condition, it will probably take them as many months to get out of it, and that, furthermore, most of the onus of responsibility will be on their own shoulders, for the examiner can only point out the road to be followed.

The feature common to all cases of disfunction is the abuse given the digestive tract. Our machine design is essentially intended for the four footed animal. A uniformly exercised and maintained musculature can compensate for the effect of gravity on the abdominal contents. The net effect of modern civilization is to permit us to depend more and more on machinery whether it be for transportation or for doing useful work, but we have not compensated by making any rational change accordingly in our diet or our body functions. "Three squares a day" are necessary for the actively working man or woman under pioneering conditions, but are certainly not indicated for those enjoying the benefits of modern civilization. These points are only possible of proof through watching the effect of less food and greater elimination on the so-called general health of patients. The fact remains however that the majority of physical examinations will show relaxed abdominal muscles, visceroptosis, and lowered intestinal tone. Exercises of various sorts are useful for the abdominal muscles, as well as for the betterment of posture which has a direct bearing on visceroptosis. Laxatives alone cannot be expected to increase and maintain smooth muscle activity. To a mild course of catharsis must be added a change of diet, increase in liquid intake and an evacuation after each meal. Any one of the

hygienic measures just enumerated, of itself, will bring no satisfactory results. They must all be put into effect at once, and in addition sufficient time must elapse before the betterment of function will even begin to be noticed by either observer or patient. Whatever changes are to ensue will be cumulative in character, and it is to shorten the beginning stage of functional inertia that mild tapering catharsis together with possible sine wave therapy are indicated in some obstinate cases. The details of these various factors must result from an intensive study of each patient as he appears. It is impossible to generalize save in the broadest sense.

In addition to digestive tract disfunction as a cause for below parness, the possibility of focal infection with resulting toxic absorption expressing itself elsewhere in the body must be kept in mind. The next most commonly encountered foci are the teeth, tonsils, sinuses and lungs. It is at once evident that the X-ray can be very useful in demonstrating the facts and it is now used as part of our routine examination. Good plates with good interpretation can tell more of value than hours of careful weighing of evidence from history taking. This line of investigation however can be held in reserve and used as may be indicated, and the cases herein outlined are of the type that are frequently encountered in the average practise, namely, the below par adult.

For the physician engaged in general practise this line of endeavor offers an interesting specialty the demand for which is on the increase, and one in which his wide and varied experience will stand him in good stead.

One point I would like to emphasize. Every patient must be regarded as an individual study. One can take nothing for granted. I have talked with many of my older medical friends about their chronic cases, and cannot help feeling that too many of the latter do not receive careful study or detailed advice. In this connection there is another pitfall that I would like to bring forward, and that is the so-called "averages." These were developed by actuarial societies for purposes other than medical. It is a mistake to assume that it is possible, let alone essential, for a poorly developed undernourished individual to approximate a greater weight such as the so-called average weight for height might be for him, providing his body functions are operating in a normal way. Similarly for the obese person who has inherited still another type of endocrine complex. We all know the limitations of diet and exercise in such cases.

From the standpoint of time, the greatest amount is spent on the physical examination which takes approximately an hour, and for which the patient is willing to pay a special fee. The discussion of the findings, together with return calls for maintenance supervision, can each

be covered in the usual half hour office visit.

I can claim no originality for this idea. There are institutions using it, national in scope of activities, which have been in existence for years. In other words the public is looking for just this sort of service, call it health examination or what you will, otherwise the institutions could not continue their existence.

To sum up, the practise of Individual Preventive Medicine as I see it today is in large measure confined to those who are dis-eased, not diseased. To render them 100% efficient functionally and able to enjoy life, our inquiry must include a complete physical examination and a thorough review of their daily lives. Questions such as cancerosis, gynecological condi-

tions, etc., can be referred for diagnosis or treatment of that particular condition, meanwhile we continue our supervision of body functions. The points most often to be straightened out revolve about diet, exercise, posture and daily habits. Of text books there are none. Our clinical experience will supply us with a fund of knowledge to enable us to start. All classes of people already appreciate the logic of keeping well, but in addition our health departments, tuberculosis associations, etc., are continually boosting the movement. The results are definite, satisfactory and lasting. I am satisfied that the practise of this specialty is practical, timely, ethical, and thoroughly in keeping with our professional altruism.

## PHYSICAL FACTORS PERTAINING TO HAY-FEVER\*

### Frequency of Seasonal Precipitation

BY A. G. GOULD, M. D.

*Assistant Professor of Hygiene, Cornell University, Ithaca, N. Y.*

Recently<sup>1</sup>, I expressed the opinion that the frequency of the rainfall must be as important as the amount of rainfall in the relief of hay-fever symptoms.

A study has been made of 26 cases representing 29 hay-fever periods, who received prophy-

frequency of seasonal rainfall on their hay-fevers.

In the previous study<sup>1</sup>, it was found that the relationship of the amount of precipitation to pollen sensitization was very complex.

Table 1 is composed of nine cases having hay-fever in the early summer, or a period roughly

TABLE 1

Early summer hay-fever cases experiencing less days of rainfall in that period of 1923 than in the corresponding period of 1922.

Case No.	Year	No. of days Rainfall	Relation 1923 amount of rain to 1922 amount	Per-cent-ages of relief
S2	1922	27		75
	1923	18	—5.37 inches	70
S4	1922	28		90
	1923	23	—3.92 inches	90
S5	1922	28		75
	1923	18	—4.69 inches	50
S6	1922	29		75
	1923	26	—5.02 inches	50
S11	1922	32		70
	1923	27	—8.53 inches	50 (a)
S13	1922	24		90
	1923	22	—6.51 inches	80
C1	1922	28		90
	1923	25	—6.18 inches	80
C6	1922	28		70
	1923	24	—8.05 inches	70
C8	1922	36		40
	1923	27	—11.83 inches	50 (b)

a—Work exposed him unduly to pollens.

b—In the hospital for surgical operation from June 16 to July 3, 1923.

lactic inoculations against hay-fever in 1922 and 1923, to determine if possible the effect of the

\*From the Department of Hygiene and Preventive Medicine, Cornell University.

TABLE 2

Late summer and early fall hay-fever cases experiencing less days of rainfall in that period of 1923 than in the corresponding period of 1922.

Case No.	Year	No. of days Rainfall	Relation 1923 amount of rain to 1922 amount	Per-cent-ages of relief
F2	1922	14		85
	1923	10	—1.66 inches	75 (a)
C2	1922	24		85
	1923	21	—3.52 inches	95
F3	1922	23		50
	1923	12	—6.82 inches	60
F6	1922	21		10
	1923	17	—6.35 inches	80
F13	1922	21		70
	1923	18	—4.81 inches	90
F24	1922	19		75
	1923	18	—0.62 inch	75
C4	1922	13		75
	1923	12	+1.30 inches	85

a—Work exposed him unduly to pollens.

from May 15th to July 15th. The number of days on which rain fell during this period of 1923 was in all cases less than in the same period of 1922. As might be expected the percentages of relief in 1923 were generally less than in 1922. Only one case gained in the percentage of relief and that individual was confined to the University Infirmary for seventeen



days of the period and was probably thereby shielded to a certain extent from the pollen. Incidentally the lessened frequency of rainfall was also accompanied by a decreased amount of rainfall varying from about four to twelve inches.

Table 2 is composed of seven cases having their hay-fevers roughly from August 15th to October 1st. During this period of 1923 these seven cases experienced less days of rainfall than in the corresponding period of 1922. Contrary to supposition these cases in general improved in their percentages of relief. In only

TABLE 3

Late summer and early fall hay-fever cases experiencing more days of rainfall in that period of 1923 than in the corresponding period of 1922.

Case No.	Year	No. of days Rainfall	Relation 1923 amount of rain to 1922 amount	Percent-ages of relief
F4	1922	21		50
	1923	24	-3.03 inches	50
F19	1922	14		50
	1923	15	-0.99 inch	75
C8	1922	18		90
	1923	22	-0.63 inch	90
F9	1922	9		50
	1923	13	-2.05 inches	80
C1	1922	16		90
	1923	21	-0.78 inch	100
F20	1922	14		70
	1923	22	+1.65 inches	85
F10	1922	13		50
	1923	15	+0.34 inch	75
C6	1922	13		zero
	1923	14	+0.44 inch	80
F11	1922	8		50
	1923	17	+3.97 inches	65
F22	1922	11		25
	1923	16	+3.40 inches	80

one case was the amount of rainfall in the season increased over the previous year's.

Table 3 consists of ten cases having hay-fever roughly between August 15th and October 1st. These cases experienced more days of rainfall in this period of 1923 than in the corresponding period of 1922, and in general improved in their relief of symptoms. In spite of the increased frequency of rainfall five of the cases had a decreased amount of rainfall in the season of 1923.

Table 4 consists of three cases having hay-fever in the late summer and early fall period mentioned above. These three cases experienced the same number of days of rainfall in their hay-fever seasons each year of the study. These cases either received the same amount of relief or an increased amount in spite of a decreased amount of rainfall in the period August 15 to October 1, 1923.

## SUMMARY

Eight cases of hay-fever experienced lessened relief with a lessened frequency of rainfall, two of these being unduly exposed to pollens.

Two cases remained stationary with a lessened frequency of rainfall and six cases experienced

increased relief, one of these being somewhat shielded by a surgical operation requiring seven-teen days' residence in a hospital.

Eight cases improved with an increased frequency of rainfall and two cases remained stationary.

I can say that sixteen of twenty-nine cases

TABLE 4

Late summer and early fall cases experiencing the same number of days of rainfall in that period of 1923 as in the corresponding period of 1922.

Case No.	Year	No. of days Rainfall	Relation 1923 amount of rain to 1922 amount	Percent-ages of relief
F5	1922	21		50
	1923	21	-5.19 inches	75
F6	1922	21		60
	1923	21	-5.19 inches	90
F16	1922	20		40
	1923	20	-2.43 inches	40

bore out the supposition that an increased frequency of rainfall was beneficial and a decreased frequency was undesirable. Six of twenty-nine cases were benefited in spite of a decreased frequency of rainfall. Five of twenty-nine cases were not affected by an increased or decreased frequency of rainfall and two of twenty-nine cases improved under the same number of days of rainfall.

Frost was not an important factor in any of these cases.

## CONCLUSION

I am not justified in saying from this study that increased frequency of rainfall is beneficial or that decreased frequency of rainfall is harmful to hay-fever cases. The facts in favor of this theory are no stronger than those against it in my series of cases. A study of a much larger series might swing the balance for or against the theory.

With the recent increased interest in the relationship of climate to disease, it is hoped that someone may be stimulated to solve the problem of the relation of rainfall and other climatic conditions to hay-fever.

## REFERENCE

- 1 Gould, A. G.: Physical Factors Pertaining to Hay-Fever. J. A. M. A., 81: 693-695, March 1, 1924.

## BOOK REVIEWS

*A Memoir of William and John Hunter.* By GEORGE C. PEACHEY. William Brendon and Son, Ltd., Plymouth, England, 1924. P. 313.

Of the many books written about the Hunters, this is perhaps the most painstaking. Dr. Peachey has already written an excellent history of St. George's Hospital, the institution which John Hunter so faithfully served for twenty-five years. He has, as he says, "lived

in an atmosphere of Hunter" for some years and certainly few men of the present era could have culled so many facts from contemporaneous literature, many of which throw new light upon the lives of the two brothers. The volume is, as the author states in the preface, "a patient and laborious compilation of facts and details." It is certainly not written for the novice in Hunterian lore, for he would soon be lost in the forest, so thick are the trees that Dr. Peachey has discovered, and yet, there is a fascination about the woods, and one never loses sight of the brothers who stand out like giants; William, the first great teacher of anatomy in England, and John, his greater brother, the founder of modern pathology.

Dr. Peachey, beginning his book as a work on John Hunter, has fortunately expanded the volume not only to include the two brothers, but also to give us a most interesting opening chapter on the early teaching of anatomy in England up to 1746. This splendid sketch leads us directly to the work of William Hunter in the Windmill Street School and to John's apprenticeship with his brother. Dr. Peachey has gained much from the current newspapers, especially in regard to dates of the beginning of lecture courses, details which have often been vaguely or carelessly set down by previous biographers.

Perhaps the most interesting chapter is that upon the last days of John Hunter. The long controversy with his colleagues at St. George's is given in great detail with the complete record of Hunter's letters and memoranda to the Board of Governors. The final scene took place on October 16, 1793. Hunter arrived late but presented his petition, urging the admission of two young men to the hospital. Someone, it is not certain who, flatly contradicted one of Hunter's remarks. "Hunter immediately ceased speaking, retired from the table, and struggling to suppress the tumult of his passion, hurried into the adjoining room, which he had scarcely reached when, with a deep groan, he fell lifeless." Dr. Peachey confines the responsibility of contradicting Hunter to two members of the Board, but is unable to choose between them. No reference of importance was made to this tragic meeting in the subsequent Minutes of the Board. "The absence of any official expression of sorrow connotes the bitterness of the hostility which he had provoked." (Peachey.)

One might call Dr. Peachey's book a book of incidentals which appear trifling, but as William Cullen said in a letter on the death of David Hume, quoted by Peachey: "To me no particulars seem trifling that relate to so great a man." To students of medical history, Dr. Peachey's book is a mine of information; to all followers of medicine, it insures delightful, if not very intimate, hours with two of the most interesting characters in all medicine.

*Controlled Diaphragmatic Breathing in the Treatment of Pulmonary Tuberculosis.* By S. ADOLPHUS KNORR, M.D.

From the fertile brain of Dr. Knopf comes this latest addition to our armamentarium against tuberculosis. In brief, Dr. Knopf believes that if the patient can be persuaded and trained to take 10-12 breaths per minute, using the diaphragm almost entirely, instead of the normal 22-24, he will be greatly benefited. He further believes that if this method is used in conjunction with other mechanical means of restricting the chest the physician's task will be made easier.

Dr. Knopf quotes from a large number of physicians in charge of sanatoria who have been polite enough to express interest in his ideas. I am strongly of the opinion, however, that no such method as this will be effective in the treatment of tuberculosis and that the whole matter is not of sufficient importance for the expenditure of any time or energy.

*Amputations.* By NORMAN T. KIRK, M.D., Major Medical Corps, U. S. Army. The Medical Interpreter. 1924. 110 pages, 35 illustrations.

This book is a comprehensive study of the amputations done in and since the World War, as well as the reamputations done on veterans. It is written sufficiently long after the war to know the results of operations done by the methods described. Such modifications of the methods as are needed for peace time surgery are suggested. Nothing but essentials are given, with the result that the book is small, concise and interesting.

The ideal stump should be of the maximum length that can be given, within certain limits. The bone end is covered by skin and subcutaneous tissue which is movable, snug enough not to crease or work into folds, free from sulci and redundancy and presenting a linear scar. Only enough muscle and fascia to form a band of scar tissue over the bone end and adherent to it is used. The stump should be painless and free from neuromata.

The following technique is given for thigh amputations. A long anterior flap with no posterior flap is used, giving a posterior scar. The anterior skin flap is dissected away from the fascia for three-fourths of an inch around its border. The quadriceps tendon is cut free from the patella. The fascia and muscles joining the tendon laterally are incised at the level of the contracted skin flaps. Posteriorly the skin flap is dissected away from the fascia; the muscles and tendons remaining are then cut through to the bone. The periosteum is cut through with a knife, the soft parts retracted and the bone cut through. A cuff of periosteum one-fourth of an inch wide is removed and the bone end rounded off. The vessels are doubly ligated. All nerves are ligated, injected with absolute alcohol and cut off. "The anterior muscle is sutured to the posterior and the fascia carefully closed. Rubber tissue drains are inserted for 24 hours and the skin is closed.

The Guillotine amputation, which was so popular in the World War, is considered to be a valuable operation for contaminated cases. By the use of traction a stump may be obtained which is longer than could be gained by other procedures. Nearly all of these cases require a secondary plastic operation to give a good stump.

Artificial limbs and other appliances have now been more or less standardized, so that better results are obtained than heretofore. Temporary appliances are used by the second or third week, before the amputee has lost his power and desire to move the limb. Temporary limbs made of fiber and costing but little are used until the stump has shrunk enough for the permanent apparatus.

**Case Records**  
of the  
**Massachusetts General Hospital**

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN  
WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY

RICHARD C. CABOT, M.D., AND HUGH CABOT, M.D.

F. M. PAINTER, A.B., ASSISTANT EDITOR

**CASE 10441**

An American clerk of sixty-six entered July 5 complaining of jaundice, abdominal distension and epigastric pain.

**F. H. Excellent.**

**P. H.** He had "variola," chickenpox, measles and whooping cough in boyhood. After that he was exceptionally healthy and strong, rarely being troubled even by colds. For four years his teeth had all been out.

**Habits.** Until six months before admission he chewed half a plug of tobacco a day. Before the prohibition law he drank about a pint of beer a day and an occasional glass of whiskey. Since prohibition he had taken no alcohol.

**P. I.** Six months before admission he awoke one morning to find himself deeply jaundiced. There was no nausea, vomiting or pain of any sort. He continued to eat, sleep and work as usual until three weeks before admission in spite of the fact that the jaundice persisted with the same intensity and for four months his skin had itched. Three weeks ago while at work he was suddenly seized with sharp low midepigastric pain, knife-like, radiating chiefly to the right upper quadrant, to a less extent to the left upper quadrant. The pain was so sharp and severe that he was obliged to sit down. The attack lasted for an hour. From the first attack to the present time there had been intermittent attacks of increasing frequency, duration and severity, lasting as long as two hours. The increase in the severity of the symptoms had been relatively little in the past three weeks. Apparently many of the earlier attacks were brought on by a meal and were frequently aggravated by calomel, which he had taken at times. For the past two weeks however he had taken nothing but buttermilk, as nothing else tasted good. Shortly after the onset of the pain he began to have gradually increasing abdominal distension. For three weeks he had been in bed, growing weaker, and had lost thirty pounds.

**P. E.** A poorly developed and very ill nourished, querulous old man whose mental processes were definitely slowed. Marked green-

yellow icterus of skin and mucous membranes, with in addition brown pigmentation of the face, neck, lower back and forearms, and numerous erosions from scratching. Skin very dry, loose and inelastic. Axillary hair absent. Breath very foul. Pain on deep inspiration. Tongue thick, furred, dry, corrugated. Some coat on gum rim and left tonsil. Lungs hyperresonant in front, even over sternum. Expansion very limited on account of diaphragmatic pain. Heart slightly enlarged to the left. Percussion difficult to right. Sounds fair. Rate slow. Frequent premature beats. Sounds at base weak. Harsh early systolic murmur over precordia, especially at the base. Marked tortuosity of the brachials and temporals, with visible pulsation. Abdomen. Flaring costal margins. Abdomen



symmetrically distended, slight shifting dullness in flanks. Liver dullness from the fourth and fifth interspaces, anterior axillary line palpable as in diagram; irregular, hard, moving with respiration, tender along the edge. Genitals not remarkable. Rectal examination. Prostate enlarged, especially to the left, not abnormally firm. Extremities. Left knee-jerk only on reinforcement, right knee-jerk lively. Some adductor contraction on attempting knee-jerk. Occasional suggestion of Chaddock and to a less extent Babinski on the left.

**T.** 97.8°-100.6°. **P.** 60-120. **R.** normal except for a terminal rise to 55. **Urine.** 3 27 at the single entry, sp. gr. 1.016, bile in very large amounts, albumin absent? **Blood.** Hgb. 100%-65%, leucocytes 15,000-24,400, polynuclears 75%, reds 4,224,000-4,040,000, no achromia or variation in size or shape. Wassermann negative. Serum dilution 1:320.

**Orders.** July 5. Fat free or low fat diet. Force fluids. Magnesium sulphate 3 i every morning. Zinc oxid wash\* by bed, to apply ad libitum. Veronal gr. x. July 7. Rectal taps 3 iv 5% glucose every four hours. Scopolamin hydrobromide gr. 1/150, repeat in an hour if necessary.

The patient showed mental aberration. He failed fast, and July 9 died.

**DISCUSSION**

BY DR. RICHARD C. CABOT

**NOTES ON THE HISTORY**

The diagnosis is almost ready-made when we have a deep jaundice at sixty-six without a previous history of anything to suggest gall-stones. It is further clinched by the painlessness of this jaundice. We have here painless jaundice in an old man for five months and a little over.

\*Zinc oxid 3 ii, calamin 3 i, glycerin 3 ii, phenol minims xx, water to make 3 viii.

We used to think that calomel was a cholagogue. Dr. Pfaff proved in this hospital that it was not, that one could not make bile flow out through a biliary fistula any faster when a patient was taking calomel than when he was not.

We start with a strong prejudice that this man has malignant disease. That is what painless jaundice at sixty-six almost always means. He has, in addition, abdominal distension and pain, making us wonder whether there is not a metastasis to produce ascites and perhaps to obstruct some portion of the gastro-intestinal tract.

#### NOTES ON THE PHYSICAL EXAMINATION

With anything wrong with the liver and brownish pigmentation we always begin to think of hemachromatosis, bronzed diabetes. This picture of the pigmentation is not characteristic; but we are not told a great deal about it. We cannot exclude hemachromatosis by anything here given.

There is nothing in the heart except that at his age and with the arteriosclerosis we may suppose he had there is associated some hypertrophy.

It is very difficult to make sure of the characteristics of the liver through the abdominal wall. I have seen a great many mistakes as checked up here. If we take these facts as true, then the probability is very large that he has cancer in the liver. We feel the irregularities of the hobnail or cirrhotic liver in only a very small percentage of cases, and the irregularities of the syphilitic liver are more gross than would be indicated by the description here. The syphilitic liver is almost cut to pieces by the bands that sink into it. The natural conclusion, if we take this record literally, is liver metastases from some tumor outside it, because primary carcinoma of the liver is so rare. We have also to remember that primary carcinoma of the liver follows cirrhosis of the hemachromatosis type, and if he has had this pigmentation for a long time, and especially if he turns out to have diabetes, we may have to think of this as secondary to hemachromatosis.

There may be pressure on the nerve roots from metastases in the abdomen sufficient to cause this abnormal increase of reflexes on one side.

The urine always contains albumin when there is much bile. I do not believe this test.

The serum dilution shows that there is about ten times as much coloring matter, presumably bile, in the serum as there should be.

#### DIFFERENTIAL DIAGNOSIS

I have very little to add to what I have said. He has in my opinion a neoplasm obstructing the biliary tract. One of the commonest places for that is the head of the pancreas. We should not necessarily get any pancreatic symptoms. We usually do not, even when there is cancer there. It can also be in the bile-ducts them-

selves, in the gall-bladder, or possibly in the intestinal tract or the liver, with metastases pressing on the bile passages. We have no evidence that it started in the stomach, but it is perfectly possible that it did. I think we really cannot go beyond saying neoplasm obstructing the biliary passages, whether within them or from without, and with probable metastases in the liver and retroperitoneal glands.

#### CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Carcinoma of the (head?) of the pancreas.  
Metastasis to liver with obstructive jaundice.

#### DR. RICHARD C. CABOT'S DIAGNOSIS

Neoplasm, probably of the head of the pancreas, obstructing the biliary passages.  
Probable metastases in the liver and retroperitoneal glands.

#### ANATOMICAL DIAGNOSIS

##### 1. Primary fatal lesion

Carcinoma of the pancreas with constriction and dilatation of the bile duct and the duct of Wirsung.  
Metastases in liver.

##### 2. Secondary or terminal lesions

Icterus.  
Ascites.  
Edema of the lungs.

##### 3. Historical landmarks

Cholelithiasis; one small stone.  
Slight chronic pleuritis, right.

DR. ALBERT E. STEELE: The liver was large, weighing 2080 grams. It showed many areas which looked like new growth. Microscopic examination of these showed adenocarcinoma.

The gall-bladder was considerably distended with bile. It contained one small stone. The cystic duct was negative. The mucosa of the gall-bladder was negative. The hepatic duct was dilated; three cm. in circumference. Its great radicles in the liver were also dilated. The mucosal surface of the upper part of the common bile-duct was smooth. The lower two and a half centimeters above the ampulla was slightly thickened and contracted by a mass of new growth in the region of the head of the pancreas. The duodenal end of the duct of Wirsung was also contracted by new growth tissue. In the pancreas it was dilated to one and a half cm. in circumference.

The tissue in the region of the head of the pancreas was pale and looked like tumor tissue. Microscopic examination showed adenocarcinoma similar to the adenocarcinoma in the liver. In other words, a cancer of the head of the pancreas which obstructed the flow of bile, with metastases in the liver.

## CASE 10442

A Nova Scotian machinist of sixty-six came to the Emergency Ward July 23, complaining of headache and dizziness of six months' duration. He was an intelligent cooperative man, on account of blindness and severe headache a good deal disoriented regarding the manner of the onset of his illness. He admitted that because of his headache his memory was poor.

F. H. His mother died of cancer of the rectum, one brother of tuberculosis of the bladder, one brother of Bright's disease (?). His wife had tuberculosis.

P. H. He had had whooping cough and mumps. At twenty-one or twenty-two he was in bed a month with rheumatic fever affecting his whole body. He did not know about redness, feet swelling, etc., but was sure he had high fever. He did fine work in a watch factory and wore a pivoting glass in the right eye, at times causing iritis, for which he went to the Eye and Ear Infirmary for treatment. For twenty-five years the sight in the right eye had been poor.

P. I. In February, six months before admission, his bowels, previously regular, gradually became constipated, and his right arm became progressively numb. Two weeks after the onset he began to have attacks of dull headache, mostly at the top of the head, accompanied by dizziness, and growing worse until at admission they were very severe, interfered with his sleep, and for five months had been practically continuous. A month or two after the onset of the headache his left eye rather suddenly became sore, red and watery. He was treated with drops by two doctors for two months with little or no relief. He then had an operation or two with the hope that the vision could be helped by iridectomy. Finally three weeks before admission the left eye was enucleated. At admission the right arm was weak, numb and at times prickly. The day before admission or at some recent period he awoke in the morning with a numb feeling in the left side of the face. On one or two recent mornings he had been unable to move his lips to speak, although he knew what he wanted to say. He had lost six pounds in six months. Since the onset of the illness he had had two attacks of vomiting within a day or two of each other following medication.

P. E. A fairly well developed and nourished old man lying rather dazed, complaining of severe headache. Left eye gone. Small supraclavicular nodule on the left. All but a few lower teeth missing. Spine fixed. Marked upper dorsal general kyphosis, said to be due to

trauma. Heart enlarged; see diagram. Position of apex not recorded. A soft systolic murmur at the apex. Sounds normal. Radials palpable. B. P. 210/110. Lungs and abdomen normal. Rectal examination. Left lobe of prostate seemed slightly large, not hard or nodular. Right pupil irregular, small, fixed; slight milky cloudiness near its periphery, perhaps in the lens. Marked arcus senilis. Reflexes. Right biceps and knee-jerk definitely greater than left. Ankle-jerk not obtained. No definite impairment of sensation. Right arm seemed weak.

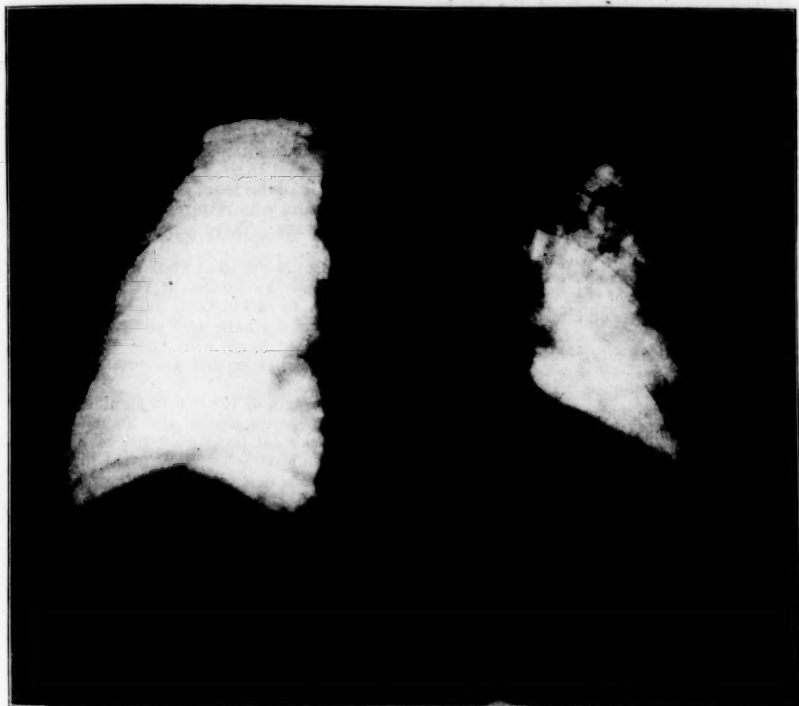
T. normal. P. 65-114. R. 17-31. Urine. Amount not recorded, sp. gr. 1.020-1.026, slightest possible trace of albumin at two of three examinations, leucocytes and occasional clumps at all of three, including a catheter specimen which also showed acetone and on culture a profuse growth of colon-like bacilli. Renal function 10%. Blood. Hgb. 80%, leucocytes 12,600-14,800, polynuclears 75%, reds normal. Non-protein nitrogen 45 mgm. Wassermann negative. Lumbar puncture July 28. 10 c.c. of clear colorless fluid. Initial pressure 90, after withdrawal of 5 c.c. 65, after withdrawal of 5 more c.c. 60. 1 cell. Alcohol, ammonium sulphate and Wassermann tests negative. Total protein 108. Goldsol 1113210000. X-ray July 25. Outline of the calvarium appeared normal. No definite changes suggesting increased pressure. No marked increase in vessel prominence. Lateral views of the skull showed in the temporal region what appeared to be several small areas of increased density simulating calcification, also some apparent change in the posterior clinoids and dorsum sellae. Some motion in these films. It was thought it would be well to repeat the examination for confirmation and further study. Owing to the patient's inability to sit up the plate of the heart was not made at seven feet, and was not satisfactory for heart measurements. Some torsion and prominence of the aortic arch. Marked narrowing of the interspace in the upper portions of both chests, probably due to kyphosis. Outline of the right diaphragm not definitely made out. There was a rounded shadow, perhaps representing the diaphragm. If so, above it there was another shadow of increased density the upper portion of which was somewhat irregular, perhaps representing thickened pleura or encapsulated fluid at the right base. July 31 the shadows previously described were present in all plates taken.

Orders. July 23. House diet without salt. Pyramidon gr. x every two hours p.r.n. Magnesium sulphate  $\frac{5}{8}$  1½ in the morning. Veronal gr. x, repeat once if necessary. July 24. To



left eye, boric acid, boric ointment, and eye wash, diluted to one half.\* Pyramidon gr. x every three hours by the clock. July 25. KI 10 drops t.i.d. July 28. Pituitrin  $\frac{1}{2}$  c.c. intramuscularly and pyramidon gr. x by mouth every three hours p.r.n.

rather little, and disappearing; later on perhaps an acute attack of pain in the eye or head. Such a case may be mistaken because of circumcorneal redness for iritis, in which case great damage may be done to the eye by the treatment for iritis, which would normally be atropin. The



Marked narrowing of the interspaces in the upper portions of both chests, probably due to kyphosis. Outline of the right diaphragm not definitely made out. There is a rounded shadow, perhaps representing the diaphragm. If so, above it there is another shadow of increased density the upper portion of which is somewhat irregular, perhaps representing thickened pleura or encapsulated fluid at the right base. Six days later the shadows

The patient became increasingly drowsy, complained less of headache, and July 31 died.

#### DISCUSSION

BY DR. MAURICE FREMONT-SMITH

#### NOTES ON THE HISTORY

The question occurs whether his headache may have been caused by the eye condition. It is possible that this man may have been developing glaucoma. In glaucoma the history is usually insidious,—several attacks of haziness of vision, perhaps pain in the eye and head amounting to

differential diagnosis rests on its finding of an enlarged pupil rather than a contracted one, a shallow anterior chamber, increased intraocular tension palpable to the fingers, and a steamy cornea. The treatment of glaucoma is eserine and not atropin. Atropin is the worst thing that can be given. In a chronic glaucoma relief can sometimes be obtained through an iridectomy or through a trephining of the cornea. It is more likely in this case that the man had a corneal ulcer which was treated for some time and which finally gave him an iridocyclitis with a hypopyon.

He was apparently not aphasic, but actually unable to move his lips.

\*Zinc sulphate gr. i. boric acid gr. xx. distilled water to make 3 i.

## NOTES ON THE PHYSICAL EXAMINATION

The heart is considerably enlarged and the supra-cardiac dullness is also somewhat increased, both of which facts may be explained by the high blood pressure. The position of the apex is not recorded. This makes us more doubtful of the actual size of the heart, the left border being less reliable than the point of maximal impulse. A soft systolic murmur is of no account. There is no doubt that the aortic second sound must have been accentuated.

The rectal examination shows at least that he does not have cancer of the prostate.

He has had frequent attacks of iritis which will explain the irregularity and the fixity of his pupil. Otherwise we should have to think of tabs. Of course in tabs the pupil would be fixed for light, but would change for accommodation.

So far we have definite evidence of some intracranial situation. We have numbness and weakness of the left side of the face and the right arm. We have the reflexes on the right increased, both knee-jerks and biceps. If we assume intracranial lesion, where must it be? Not in the cerebral cortex, because in that case if we had a facial paralysis and an arm and leg reflex change the two would have to be in the same side. A lesion on one side of the cortex would give face and arm and leg on the opposite side. The same is true of a lesion in the subcortex or internal capsule. We have to go down to the region where the facial nerve on the opposite side can be involved directly. That could be caused either by pressure from a tumor of the cerebellum or one at the cerebellopontile angle. We have not enough examination here to rule out a cerebellar tumor. We should want to know about nystagmus, about adiadochokinesia, about his gait, muscle tone, etc., all of which things we probably could not get in this case. With regard to tumors in the cerebellopontile angle, usually these tumors show early involvement of the eighth nerve with deafness and tinnitus; then as the size increases other cranial nerves are involved, the sixth, fourth and fifth sometimes. In any case we have however to assume a lesion at the base of the brain. More than that I think we cannot say at this moment.

He had a bacilluria and probably had some low-grade infection of the kidneys.

This is the sort of renal function that one would like repeated. If repeated at ten per cent. of course there must be definite renal pathology. It takes a very definite amount of renal pathology, unless it be in a case with chronic retention as in an obstructing prostate, to give as low a function as ten per cent.

MISS PAINTER: Here it was done twice and was ten per cent. each time.

DR. FREMONT-SMITH: A hemoglobin of eighty per cent. in itself is against a chronic nephritis

as such. The leucocyte count was done probably toward the end.

I think we had a right to expect an increased intracranial pressure, and here we find an initial pressure in the spinal fluid of ninety, which is low. It is possible that our original assumption is wrong. On the other hand it is possible that there may be a block in the region of the fourth ventricle, in which case we might have an internal hydrocephalus with a very high pressure, and a low pressure in the spinal fluid. There is one statement that we should like to have, and that is what happened on jugular compression and on deep breathing. If we find that there is a considerable rise in pressure on compression we can be sure that there is no block in the fourth ventricle. Likewise there is normally a rise and fall of pressure with respiration.

MISS PAINTER: There is no record of jugular compression.

DR. FREMONT-SMITH: That is something we should have. We cannot at this moment say with regard to intracranial pressure, and that simple test would have been sufficient to differentiate between block with possible high pressure in the ventricles and a normal and rather low-pressure fluid.

A total protein of 108 is very significant. Here we have a negative alcohol and ammonium sulphate, but a total protein of 108 as against a normal of thirty-five to forty in the lumbar region (and of about seven mgm. in the ventricles). In other words this one finding of increased total protein makes it possible to say that there is definite pathology in the central nervous system. The goldsol is of abnormal curve, although it is not specific for any disease-entity. In one hundred normal gold tests there will be perhaps ninety all zeros, and ten with a few ones, but a two is usually abnormal, and three is always definitely abnormal.

As a matter of fact oftentimes the first evidence in the calvarium of increased pressure is absorption of the posterior clinoids, so this X-ray does not rule out increased pressure. And sometimes one may have increased pressure with no evidence at all by X-ray.

The orders are not important except that I should like to know why pituitrin was given.

## DIFFERENTIAL DIAGNOSIS

There is one possibility to consider which may be the answer here. In the first place this situation is atypical, because physical examination—either because of insufficient physical examination or the sickness of the patient or because the actual condition did not exist—did not correspond to the patient's symptoms. He complains of tingling and lack of sensation in his right hand and arm and a numb feeling in the left side of the face. These sensory disturbances are not confirmed by physical examination. We

should like to know about the examination as regards heat and cold. At any rate the picture is atypical. We do not find enough. Here is a man who has a progressive intracranial lesion. Physical examination gives us few positive signs. His first symptom was that of constipation. He had a supraclavicular node on the left. He is a man of sixty-six. He has lost some weight. We must consider the possibility of carcinoma with metastasis to the brain, and I had hoped to say to the chest too. Dr. Cabot, can you give any interpretation of that shadow that would be satisfactory to you?

DR. CABOT: No. I do not see any evidence of metastasis, and I have never seen anything like that diaphragm shadow. My guess is that these are adhesions, as they said, and that nothing special will be found.

DR. FREMONT-SMITH: My feeling is that if we are allowed to examine the head by necropsy we shall find a lesion, probably a metastasis below the tentorium at the base of the skull, also metastasis to the node above the clavicle. The other possibilities are tumor, old hemorrhage with necrosis, or thrombosis with softening. We shall find the other evidence of hypertensive heart disease, a big heart, arteriosclerosis, arteriosclerosis of the kidneys. What we shall find in the chest I do not know.

A PHYSICIAN: Do you think a cerebral arteriosclerosis in itself could explain those symptoms?

DR. FREMONT-SMITH: That is something I should have spoken of. One can have atypical symptoms from arteriosclerosis, but this seems to me too definitely localized for that.

#### X-RAY INTERPRETATION

Findings suggestive of suprasellar cyst.

#### CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Cerebral arteriosclerosis.  
Brain tumor?

#### DR. MAURICE FREMONT-SMITH'S DIAGNOSIS

Malignant disease with metastases to the brain.  
Arteriosclerosis.  
Arteriosclerotic degeneration of the kidneys.  
Hypertrophy and dilatation of the heart.

#### ANATOMICAL DIAGNOSIS

##### 1. Primary fatal lesions

Arteriosclerosis.  
Arteriosclerosis of the vessels of Willis with partial occlusion of the basilar artery.  
Area of softening in the right cerebellar hemisphere.

##### 2. Secondary or terminal lesions

Wet brain.  
Edema of the lungs.

Hypertrophy and dilatation of the heart.  
Fibrous myocarditis.  
Congestion of the liver, spleen and kidneys.

##### 3. Historical landmarks

Hypertrophy of the middle lobe of the prostate.  
Diverticula in the urinary bladder.  
Obsolete focus of tuberculosis of the right lung.  
Chronic pleuritis.

DR. RICHARDSON: The pia showed marked infiltration with thin pale fluid, and there was some fluid at the base. The vessels of Willis and their branches near and remote showed marked arteriosclerosis. In the basilar artery there was marked arteriosclerotic degeneration and a few thrombi adherent to the wall in the situation of the sclerosis, causing partial occlusion. With that there was an associated area of softening in the posterior half of the right cerebellar hemisphere.

In the background of this case there was of course arteriosclerosis. We found arteriosclerosis of the aorta and great branches. The heart showed considerable hypertrophy and dilatation. The valves were negative. The myocardium showed a few areas of fibrous myocarditis.

The liver, spleen and kidneys showed chronic passive congestion, and there was some arteriosclerosis of the vessels of the kidneys. The bladder was markedly distended, and in the situation of the so-called middle lobe there was an ovoid mass which considerably obstructed the urethra. The trabeculae of the bladder were hypertrophied, and there were a few diverticula between these trabeculae. This is rather interesting as illustrating Dr. Fremont-Smith's remark about this condition.

There were pleural adhesions on the right, one band in the region of the upper lobe.

DR. CABOT: The shadow in the X-ray plate remains a mystery.

A PHYSICIAN: What about the discrepancy between the kidney function and the specific gravity of 1.025 to 1.028?

DR. CABOT: Under those conditions I always go for the gravity. I have seen ten per cent. before with kidneys essentially normal. I think it has to be below ten before we can make any bets on nephritis.

#### CASE 10443

An Irish fireman of forty-five entered September 13 complaining of blindness of the right eye and deafness of the right ear.

F. H. Good so far as known, except that his wife had had one miscarriage and one of fifteen children stillborn.

P. H. His general health had been good. He had measles in childhood. He had had some shortness of breath on exertion, and occasional attacks of "indigestion" with epigastric distress. He urinated seven to eight times by day and once or twice at night.

P. I. A year before admission he began to notice slight impairment of the hearing in the right ear. Seven months later, in the April before admission, he became dizzy on bending over and occasionally lost his balance, especially if while bending his head he kept his eyes closed. Soon after this he developed headache which persisted until four weeks before admission, when it became a negligible feature. In May he became conscious of gradual loss of visual acuity in the right eye. This increased until at admission he could not see at all with this eye. In the past two months the left eye had weakened considerably. His distant vision was fairly good, but he could not see objects near to him. He had recently had slight dysphagia which he attributed to his apprehension over his condition. He was able to continue his routine of life until four weeks before admission, when his failing vision had caused him to stop work. He had lost twelve pounds since spring.

P. E. A well nourished man who gave a concise history. Right pupil slightly smaller than left, and right conjunctiva injected. Slight right internal strabismus. Right ear deaf. Pyorrhea. Heart not enlarged. A<sub>2</sub> accentuated. B. P. 210/140. Lungs, abdomen, genitals, extremities and pupillary reactions normal. Rectal examination. Prostate slightly enlarged and indurated. Eye grounds. Almost total blindness of right eye. Abnormally constricted in left eye. Choked disc, moderate degree, both eyes. Reflexes. Patellars active. No Babinski or clonus. Scrotal and abdominal reflexes present. Romberg unsteady; patient inclined to sway to the right. Note by Dr. Mixer, "I find in addition a right facial weakness and choked disc. This gives us sixth, seventh and eighth, with dizziness and tinnitus . . ."

T. 98.1°-99.1°. P. 121-80. R. 20-24. Urine. Amount not recorded, sp. gr. 1.008, cloudy at the single examination, no albumin or sugar. Ear consultation. "Total deafness right ear, normal hearing left ear. Bárány, caloric, water at 68. Summary. No response from right ear or left vertical canals, normal responses from left vertical and horizontal canals. Spontaneous inward past-pointing with left arm. Findings suggestive of a right cerebellopontile angle lesion causing enough mesial pressure to affect the vertical canal fibers of the left side." Medical consultation. "I can find no definite evidence of a cause for hypertension other than the cerebral. I know no way of satisfactorily reducing pressure, and furthermore I do not believe the pressure here makes operation particularly more dangerous. I should expect that

the operation itself would bring the pressure down materially."

September 18 operation was done. The patient died on the operating table.

#### DISCUSSION

BY DR. W. JASON MIXTER

Loss of vision, dizziness associated with deafness in the right ear but without headache, while not conclusive we must consider indicative of brain tumor or some other cause of increased intracranial tension as one of the probabilities in this case.

Definite evidence of increased intracranial tension as shown by choked discs, total deafness of the right ear, right facial weakness, would point to a lesion in the right cerebellopontile angle arising from the eighth nerve, from the dura, or possibly from the cerebellum. There is no reason to suspect an inflammatory process such as abscess or tuberculosis.

#### CONCLUSION OF NEUROLOGIST SEPTEMBER 13

"Caloric tests done in Nerve Department August 31. Water 64°. Right ear douched. All responses cut off. No vertigo. No past-pointing. No nausea or nystagmus. Indicating right sided lesion in pons."

#### CONCLUSION OF SECOND NEUROLOGIST SEPTEMBER 15

"Lesion should be in right cerebellar fossa. I believe it to be an acoustic neuroma. Early operation advised."

#### DR. MIXTER'S PRE-OPERATIVE DIAGNOSIS

Probably right acoustic neuroma.

#### PRE-OPERATIVE DIAGNOSIS

Acoustic neuroma, right.

#### OPERATION

Gas and ether. Cross bow incision over occiput carried down to the skull at once. A few vessels in the skull were clamped, and then clamps were placed to the galea, which was then pulled up over the skin. The bleeding was well controlled. The incision was carried down in the midline to the atlas, and flaps were turned downward and outward. The bone was cleaned with periosteal elevators. Bone removed over cerebellum from lateral sinus downward to and including the posterior third of the foramen magnum. Posterior portion of the arch of the atlas was also removed. The right ventricle was tapped through a drill hole in the right occipital region, as the dura was tense and bulging, relieving the pressure. A T-shaped incision was made in the dura. This exposed the medulla and cerebellum, which showed a pressure cone. The right lobe of the cerebellum was retracted upward disclosing the seventh nerve

and the tumor under it. At this point the posterior inferior cerebellar artery was cut and had to be clipped. Removal of the tumor by blunt dissection was attempted, but a large part of it lay in front of the medulla and could not be dislodged. Finally a finger was introduced external to and above the tumor, which was then bulging upward and outward, and the tumor drawn out. There was a large gush of blood and the patient stopped breathing. A sponge was hastily stuffed into the cavity. The patient was placed on his back with a towel over the incision, and artificial respiration was begun. This was continued for an hour and a half, at the end of which time no signs of spontaneous respiration had appeared. The patient was turned on his face again with fresh drapings and instruments. The sponge was removed. The bleeding had mostly stopped. Artificial respiration was continued. The wound was closed; but the heart stopped beating while the last sutures were being put in.

#### **PATHOLOGICAL REPORT**

A soft spherical tumor measuring  $4\frac{1}{2}$  cm. in diameter and showing a purplish-red surface on section with pale yellowish areas.

Microscopic examination shows interlacing bundles of elongated spindle cells with fluid and mononuclear wandering cells between their fibrils. There are collections of pigment-holding cells and masses of granular pigment.

Acoustic neurofibroma.

#### **FURTHER DISCUSSION**

The cause of death in this case was evidently disturbance of the respiratory center when the tumor was dislodged from in front of the medulla. On account of the bleeding pressure had to be maintained in the attempt to check it, and this pressure undoubtedly prevented the respiratory center from taking up its function. In the light of more recent knowledge it is probable that today no attempt would be made to enucleate such a tumor. Incision of the capsule and evacuation of the tumor is a much less dangerous method of approach. This case presents rather unusually clear cut localizing signs of cerebellopontile angle lesion, and shows the danger of radical enucleation where the new growth lies in front of the pons and medulla.

#### **CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)**

Acoustic neuroma.  
Respiratory paralysis.  
Excision acoustic neuroma.

#### **DR. W. JASON MIXTER'S DIAGNOSIS**

Acoustic neurofibroma.

#### **ANATOMICAL DIAGNOSIS**

(Acoustic neurofibroma of the right cerebellopontile angle.)

Operation wound in the occipital region, craniotomy.  
Hemorrhage in the region of the right cerebellum.

DR. RICHARDSON: The head only was examined. The brain weighed 1612 grams. There was marked flattening of the cerebral convolutions. The cerebral meninges were negative. At the base of the brain beneath the operation wound there was considerable blood and blood clot. The right lobe of the cerebellum showed considerable maceration. The brain tissue elsewhere showed nothing of note. The sinuses and middle ears were negative.

### **CURRENT LITERATURE**

#### **ABSTRACTORS**

GERARDO M. BALBONI	TRACY MALLOY
WILLIAM B. BREED	HERMAN A. OSGOOD
LAURENCE D. CHAPIN	FRANCIS W. PALFREY
AUSTIN W. CHEEVER	EDWARD H. RISLEY
RANDALL CLIFFORD	GEORGE C. SHATTUCK
ERNEST M. DALAND	WILLIAM H. SHEDDEN
HORACE GRAY	WARREN R. Sisson
ROBERT M. GREEN	JOHN B. SWIFT, JR.
GEORGE B. HAWES, 2ND	JOHN C. SMITH
JOHN S. HODGSON	W. T. SHERMAN THORNDIKE
FRED S. HOPKINS	WILDER TELESTON
CHESTER M. JONES	HENRY R. VIETS
CHARLES D. LAWRENCE	SHIELDS WARREN
	BRYANT D. WETHERELL

#### **THE ROLE OF LIGAMENTOUS CALCIFICATION IN LOWER BACK PAIN**

DOUB, HOWARD P. (*The American Journal of Roentgenology*, Vol. XII, No. II, p. 168).

The ilio-lumbar ligament is a powerful, flat fiber band which has its origin from the transverse process of the fifth lumbar vertebra and extends outward to the surface of the iliac crest. These ligaments when seen on the film are triangular in shape, with the base of the triangle continuous with the transverse process of the fifth lumbar vertebra, and the apex of the triangle being at the insertion of this ligament along the crest of the ilium. Calcification occurs not only in this ligament but also at a new area of bone formation on the crest of the ilium at the point of insertion. This small projection may be seen in many cases in which the shadow of the ligament itself cannot be demonstrated. The majority of the cases have shown evidence of infectious arthritis varying from a slight spur formation to calcification of all the ligaments of the spine. It is the opinion of the author that this condition is a manifestation of infectious or hypertrophic arthritis and not a distinct entity. He reports one case where surgical removal of the involved ligament offered complete relief.

[H. A. O.]

#### **TREATMENT OF TUBERCULOSIS WITH ARTIFICIAL LIGHT**

WESSELY reports (*Wien. klin. Woch.*, June 19, 1924) a new method of treating tuberculosis of the upper air passages by locally applied artificial light. He describes his technic and regards it as particularly applicable to laryngeal tuberculosis.

[R. M. G.]



## THE BOSTON Medical and Surgical Journal

Established in 1828

Published by The Massachusetts Medical Society under the jurisdiction of the following-named committee:

For three years JAMES S. STONE, M.D.  
HORACE D. ARNOLO, M. D.  
CHANNING FROTHINGHAM, M. D.  
For two years HOMER GAGE, M.D., *Chairman*.  
EDWARD C. STREETER, M. D.  
EDWARD W. TAYLOR, M. D.  
For one year WILLIAM H. ROBEY, JR., M.D.  
ROGER I. LEE, M. D.  
ROBERT H. OSGOOD, M. D.

### EDITORIAL STAFF

DAVID L. EDGALL, M. D.  
WALTER B. CANNON, M. D.  
RED HUNT, M. D.  
\*ROBERT W. LOVETT, M. D.  
FRANCIS W. PEARSON, M. D.  
JOHN P. SUTHERLAND, M. D.  
S. BERT VOLZACH, M. D.  
GEORGE R. MINOT, M. D.  
FRANK H. LAHEY, M. D.  
STEPHEN RUSHMORE, M. D.

WALTER P. BOWERS, M. D., *Managing Editor*

### ASSOCIATE EDITORS

GEORGE G. SMITH, M. D.  
WILLIAM B. FRENCH, M. D.  
JOSEPH GARLAND, M. D.  
\*Deceased.

SUBSCRIPTION TERMS: \$6.00 per year in advance, postage paid for the United States, \$7.50 per year for all foreign countries belonging to the Postal Union.

Material for early publication should be received not later than noon on Saturday. Orders for reprints must be sent to the printer with galley proof of paper.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to The Boston Medical and Surgical Journal, 126 Massachusetts Ave., Boston, Mass.

### REGISTRATION OF MIDWIVES

THE Recess Committee of the Legislature, Senator Draper, Chairman, conducted a hearing Oct. 15 based on House Bill No. 223 introduced last winter by Mr. Graves of Springfield.

The proponents represented the Springfield Woman's Club; the Social Service organization of Springfield; Miss McCrea of Fall River; The Massachusetts Civic League; The State Federation of Women's Clubs; Miss Donaldson, a member of the Legislature; the Woman's Civic League of Brookline, and others.

This movement has been in progress for the past six years and is based on the belief that there are many incompetent midwives in this state, that there is a demand for well educated midwives in many communities and that doctors do not meet the needs of those people who have been accustomed to employing midwives. The abuses at present are claimed to consist of dangers incident to unskilled or meddling midwifery which are not always confined to midwives.

Dr. Frederick R. Williams of Worcester made some startling assertions. He claimed that the competition among doctors in Worcester is so

keen that physicians doing a large obstetric practice and especially some working among the poorer classes do not give adequate time to women in labor, in that operative interference is resorted to too early in order to save time, with a high mortality rate, and further, that this rate is rising; also, that some doctors are in collusion with midwives, charging three dollars for reporting births attended by midwives and also collecting the legal fee of twenty-five cents. Referring to the midwives, he stated that the Board of Health in that city inspects the bags used by midwives so as to eliminate the use of instruments and virtually endorses those midwives who have an equipment that passes the inspection. Midwives when presenting bags for inspection leave out the hypodermic syringe and other instruments but use pituitrin subcutaneously and perhaps other drugs and even instruments. He felt sure that there are many technical errors in this practice. On cross examination by the Committee he admitted that his criticisms of doctors apply to a minority. He felt that the number of midwives in Worcester is increasing but could not give actual figures.

Proponents from Springfield and Fall River also said that doctors were returning births attended by midwives for a fee of three to five dollars.

A high stillbirth rate was reported from Fall River which, in the opinion of the reporter, could have been modified if skilled service had been given. Eighty-four stillbirths attended by midwives were reported by medical men. Other cases of low-grade service were reported.

Drs. Charles A. Mongan of Somerville and W. P. Bowers of the MEDICAL JOURNAL opposed the registration of midwives on the ground that since the Supreme Court had rendered an opinion to the effect that the practice of midwifery is the practice of medicine that legal registration of midwives would be contrary to the state policy in that it would create different standards of practice, and further, that the pregnant women in one city or town are entitled to receive the same quality of service as those in other localities.

Of the midwives in Massachusetts probably not over five per cent. could qualify before the most lenient examining board and this small number would not solve the problem. The opponents felt that no sufficient argument had been advanced to warrant the adoption of the plan to register midwives.

Dr. Mongan severely criticised those who are in collusion with midwives in maintaining illegal practice. Testimony of this custom ought to lead to legal action for the law provides that any physician who associates himself with an unregistered person for the purpose of carrying on the practice of medicine is open to prosecution and may be fined or suffer loss of his registration.

Now that these assertions have been made in public it is clearly the duty of the Board of Registration in Medicine to call those cognizant of this practice before it and if the testimony is confirmed to proceed against those physicians involved in this illegal practice.

It seemed to be evident that the committee conducting this hearing feels deeply concerned about the problems involved.

### ETHER DAY AT THE MASSACHUSETTS GENERAL HOSPITAL

THE great experiment of securing insensibility by the use of sulphuric ether in preparation for human surgery was again fittingly commemorated at the Massachusetts General Hospital, October 16, 1924.

The exercises consisted of demonstrations in the surgical amphitheatre during the forenoon and a meeting in the afternoon to hear Professor Zinsser. The morning program was as follows: "Remarks on Asthma" by Dr. F. M. Rackemann; "Bone Tumors" by Dr. C. C. Simmons; "Acute Dilatation of the Stomach" by Dr. M. A. McIver; "Urinary Calculus" by J. D. Barney; "Results of Tryparsamide Treatment" by Dr. H. R. Viets; "Rational of the Use of Glucose in Treatment of Acidosis" by Dr. F. B. Talbot; "Surgery in Diabetes" by Dr. D. F. Jones; "Treatment of Lead Poisoning" by Dr. J. C. Aub and "Lipidol in the Diagnosis of Spinal Block" by Dr. W. J. Mixter. An operative clinic was held from 12:00 to 1:00 o'clock, after which luncheon was served in the brick corridor.

At three o'clock the exercises were resumed in the assembly room in the Moseley building.

Dr. Henry P. Walcott called the meeting to order and introduced Dr. Frederic A. Washburn, who reported the names of the graduate house officers as follows:

House Officers graduated, October 16, 1923, to October 16, 1924:

*Medical*—Thomas Maxfield Barber, East Medical; Eugene Adolph Osius, West Medical; Paul Howard Means, East Medical; Louis Wolff, West Medical; Howard Burnham Sprague, East Medical; George Colket Caner, West Medical; Walter Wendell Fray, East Medical; Michael Lake, West Medical.

*Surgical*—William Eichelbert McConnell, East Surgical; Stephen George James, West Surgical; Francis McTear Pindlay, East Surgical; Robert Leonard Mason, West Surgical; William Franz Harper, East Surgical; Grantley Walden Taylor, West Surgical; Edward Joseph Ottenheimer, East Surgical; James Doney Bisgard, West Surgical; Thomas Banford Jones, Third Surgical.

*Children's Medical*—Benjamin Brock, Erastus Ingham Guller, Siegfried Eliasoff Katz, LeRoy Franklin Cotterson.

*Orthopaedic*—Lemuel David Smith, James William Martin, Thomas Ray Haig, Hervey Heyward Wescott, William Alexander Rogers.

Number of living alumni:

Graduate house officers..... 609

Active members of M. G. H. staff, non-graduates..... 124

Dr. Walcott then gave an eloquent eulogy of William T. G. Morton and in introducing Professor Zinsser outlined certain features of the progress of medicine, paying especial reference to bacteriology.

Professor Zinsser's address was an elaborate portrayal of the advances of medicine and the responsibilities of physicians. The social and political problems which must be met were referred to and advice given as to the proper attitude of the profession. He explained some of the psychological reactions of the laity in meeting the arguments of physicians and recommended tolerance and even generous treatment of those who are unable to understand the purposes of advocates of restrictive legislative measures in handling public health problems.

We are to have the honor of publishing this address and assure our readers that it should be carefully studied.

### THE SMOKE NUISANCE

EVERY large city and many smaller manufacturing communities suffer from excessive smoke. Anything which obscures sunlight and contaminates the air may affect health. Smoke is also a financial burden because it necessitates more time and expense in keeping clothing and dwellings clean and represents fuel waste.

Large cities are paying especial attention to the problem of diminishing the amount of smoke.

With characteristic energy Chicago has made a definite campaign against the smoke nuisance through transfer of the enforcement of the smoke ordinance to the Department of Health. The known violations of the ordinance in this city amounted to 1468 cases in 1918, reaching the figures of 8284 in 1920. In 1918, 68 chimneys out of every 100 observed were violating the ordinance, but in 1924 only 20 per cent. were at fault.

Including fuel waste, as well as direct damage, it has been estimated that smoke has caused a loss of \$42,500,000 a year in Chicago, and the irritation of the air passages is supposed to favor the development of respiratory diseases and may have a definite effect in increasing susceptibility to tuberculosis.

Chicago's example has led to the estimate that there are probably 16,000,000 amateurs managing fuel consuming plants in the United States with a great financial loss. This means that even the small consumer of fuel may do his part toward lessening the smoke nuisance as well as saving money. He should be ambitious to aid in health maintenance as well as to practice economy.

Every person concerned is under obligation to meet the needs of the community. When health departments generally pay attention to this matter more progress will be made throughout the country.

## CORRESPONDENCE

## LONDON LETTER

(From Our Regular Correspondent)

## DEATH OF SIR WILLIAM BAYLISS

The greatest, perhaps, of modern physiologists died in London, recently, in the person of Sir William Maddock Bayliss, Professor of General Physiology in University College.

Born in Wolverhampton in 1866, he was for a short time engaged in the business of his father, who was an iron manufacturer. He soon found this work uncongenial and took up the study of medicine, which also he abandoned—at least temporarily. His bent was for pure science and he became a student of University College, London. He took his B.Sc. degree there in 1882 and the University Scholarship in Zoology, and again reverted to medicine, studying physiology first under Burdon Sanderson, and then, when the latter left for Oxford, under Schafer. However, as said before, he was always more greatly attracted by the purely scientific side of his studies and took but a languid interest in the dry bones of anatomy, in which subject he failed in his second M.B. examination.

Deciding that further work in a branch of medicine in which he took no interest was not worth while, he finally gave up the idea of a medical qualification and turned his whole attention to physiology. He therefore followed his beloved teacher, Sanderson, to Oxford, entering at Hadham College in 1885, and in the Final School of Natural Sciences in Physiology was awarded a first class. He was endowed with an insatiate thirst for information, and, being considerably in advance of the students of his year in general science as in special subjects, could afford the time to slake this thirst and delve into art, literature, science and philosophy. He thus acquired that catholicity of taste and breadth of learning which ever distinguished him in later years among his compeers.

When he had taken his degree he returned to London, to live in a house which his father had built on the slopes of Hampstead Heath, the most picturesque suburb of London. He at once took up work again at University College under Schafer.

G. H. Starling, in 1890, left Guy's Hospital, where he had been teaching physiology, because he was unable to obtain facilities for research, came to University College and joined hands with Bayliss. Thus was begun that remarkable scientific partnership which brought forth some epoch-making discoveries on the electrical phenomena of the heart, the action of the nerves on the heart, the regulation of the circulation, the movements of the intestine, pancreatic secretion, and the action of hormones. Bayliss also carried out researches on vaso-motor nerves with John Rose Bradford, and on the circulation of the brain with Leonard Hill.

In 1893 he published his classical paper on the depressor nerve, and a series of papers on vaso-dilator nerves, their origin and mode of action, published between 1901 and 1908.

In 1909 Starling was appointed to the Jodrell Chair of Physiology at University College, and Bayliss came to his aid, first as assistant and then as assistant professor, until finally, as Professor of General Physiology, he took an active part in the teaching work of the department. This direct contact with the students appeared to greatly stimulate his mental activity, as from 1904 up to the time of his death almost, a long stream of papers and monographs flowed forth, which dealt especially with physico-chemical problems of physiology and with the mode and action of enzymes.

In 1914 he brought out his magnum opus, that masterly work known as "The Principles of General Physiology." This work is in a way sui generis as

it not only discusses science but discloses the personality of the author. It is a history of a mind and its workings and its achievements. The preface to the first edition gives an idea of the man and of the wide range of his vision and interests, in which he lays down his views of the conceptions of physiology and how these may be obtained.

The success of this book was striking and wide spread, and it was hailed by workers in all sciences as the very embodiment of the living spirit of physiology. As a proof of the high value placed upon it, two Bayliss Societies have been founded in America, at meetings of which its members come together at regular intervals to read and discuss the several chapters of the book.

But an even more remarkable proof of the estimation in which the book was held and the love felt for the writer was that Bayliss was a prophet with honor in his own country. When the preparation of a fourth edition was hindered by the illness of the author the work was undertaken by a number of young physiologists under the editorship of Leonard Hill, each taking the chapter the subject matter of which he knew the most, with the object of doing homage to the man and of placing at the disposal of scientific workers a book which had proved a fruitful source of instruction and inspiration.

During the war Bayliss gave special attention to wound shock and devised a mode of treating the condition with gum saline, which was widely used and met with a considerable amount of success.

The extent and importance of his scientific work may be gauged by the long list of distinctions conferred upon him. These included: Member of the Council of the Royal Society; president of the Physiological Section of the British Association; treasurer of the Physiological Society and editor of *Physiological Abstracts* and the *Biochemical Journal*; Fellow of University College, London; Honorary Fellow of Wadham College, Oxford; Croonian Lecturer, Royal Society; Royal Medal, Royal Society; Copley Medal, Royal Society; Baby Medal, Royal College of Physicians; Oliver Sharpey Lecturer; Silvanus Thompson Lecturer; Herter Lecturer; chairman of the Shock Committee of the Medical Research Council and the Committee on the Biological Action of Light; member of the Chemical Warfare Medical Committee and of the Research Committee of the Food Investigation Board.

In 1893 Bayliss married Gertrude Starling, sister of his friend and fellow worker.

Bayliss was a unique character. He was the foremost physiologist, and a research worker and philosopher of the widest intellectual range. He was a modest, unassuming man and bore his great knowledge with befitting humility.

## PROBLEMS INCIDENT TO PROHIBITION

Mr. Editor:

With the advent of prohibition—rather, the adoption of the Eighteenth Amendment—it was supposed that the use of intoxicating liquors as a beverage would diminish so that drunkenness would become a rare condition. It was said the next generation would hardly know the taste of alcoholic liquors or the sight of a drunken man. The jails were to close because of lack of inmates—an acknowledgment of the close association between alcoholism and crime. The confirmed drunkard and inebriate would be a thing of the past, and the sanitaria and homes for his treatment and attempted cure would be obliged to take up some other line of endeavor or close their doors.

What has happened since the passing of the Act of Congress of October 28, 1919, known as the National Prohibition Act? Alcoholism is still with us, and inebriety, as some of us observe it, is not diminishing numerically or in severity. Prohibition has closed the wide-open saloon but has not dried up the source

of supply of alcohol as a beverage, so that to those who have the care or treatment of such cases prohibition is apparently a failure. I am not a believer in prohibition. In the first place, it does not prohibit, and without adequate enforcement it is a farce. Alcoholic liquor can be bought in numerous places, known to many, these places doing business openly. The proprietors of such places are strong advocates of prohibition, as upon it depends their business with its present large profits. When the time comes that the proper authorities can or will act in such cases it will be worth while to report them, but at present there is nothing gained, besides which it is hard to believe that officers of the law do not know of places that are commonly known to the public, where liquor is being sold. Prohibition has not benefited those who need it most. The cost of the liquor has increased and the quality become poorer. Former beer drinkers have become "hooch" drinkers, as they can buy a drink over the bar, as well as by the bottle, and cannot get the less harmful beer and light wines of former days. This condition is not local but general. With what is being smuggled in plus what is being illicitly distilled, the supply is apparently inexhaustible.

This being the condition as at present existing, the disease condition known as inebriety still deserves our consideration, and my own experience is that this disease is increasing under the Volstead Act. The treatment, being essentially institutional, should still be provided for. As seen in private practice in men of the intellectual type mostly, and in old as well as new cases, it is noticeable that over a period of years the subject of this disease has suffered impaired health and impaired will power. Lowered resistance makes them especially susceptible to other disease, especially tuberculosis. The general character suffers a moral decay, and especially is there a noticeable lack of truthfulness. Even the better educated gradually lose their ambition and self-respect and with decreasing will power they become more and more degraded. At times he seems to become aware of his condition, and I have known them to show the greatest confidence in their ability to get the better of it, but they never want to begin then, so they go from bad to worse. I believe in the prolonged treatment of these cases, which I will speak of later. It is sometimes surprising, when looking into the history of a common drunk, to find that he came from a good family and had a good education. A family history frequently shows a like tendency in parents or other not very remote relatives. There seems to be no question as to hereditary tendency. Where this is known the parents should give the child proper care and guidance in early youth, for education and environment, healthful living with outdoor exercise and proper moral and religious atmosphere, at an early age, will help to overcome the tendency.

Taken later in life, treatment is discouraging. Much, however, can be done, and if the patient, or subject, will submit voluntarily to treatment, the attempt is worth while. At the present time there are places where such cases can go and "sober off," this process taking from a few days to a week. He goes out sober, but not cured. In some cases he still has the craving, which he at once proceeds to satisfy, while in other cases, the distinctly hereditary cases of inebriety, he has no desire and may remain sober for from one to six months. The moral decay and dulled sensibilities, with the resulting undermined character, sooner or later bring about another fall and his will power is still further weakened. He intended to give up the habit of drink, but he has postponed it once more. Physically, mentally, morally, spiritually, he is weaker after each attack, and while this is not apparent to him it is noticed by the family.

How are we to treat these cases? The common

drunk can go to an institution and sober off, the chances being that he will be drunk again soon. The inebriate also sobers off and usually goes out without the desire for a drink, but he is not cured by a few days in a sanitarium or institution. The inebriate should submit voluntarily to treatment in an institution for that purpose for a period of from two to four months. The place must be somewhat homelike and the surroundings pleasant and agreeable. Such a place, in or close by a large city, should have opportunity for inmates to work in the garden or for outdoor exercise. Fresh air and sunshine are important. We are simply to try and assist nature and keep the patient within bounds where he may be protected from himself. Medical supervision with care of the general health, diet, hours of sleep, exercise, recreation, visitors, all to be supervised and the patient made to feel that a real human interest is taken in him, will all tend to reestablish and strengthen his will power and confidence in himself. Such a place would not partake of the nature of a jail, although freedom would be restricted to some extent. A real human interest in each individual case is needed. Drugs play but a small part in the treatment. It is true that few places are properly located or arranged for the ideal treatment; but much might be done with such facilities as they have. In some cases they have no funds to spend for improving their conditions or surroundings. The high cost of living and of labor have affected them seriously, especially where they have a fixed income from investments. In such instances the proper treatment of these cases might be helpful alike to patient and institution, as the increased revenue from these cases would make it possible to improve the standard of the institution.

No cure can ever be guaranteed, but that treatment briefly outlined above is the most hopeful of results. As these cases are apparently to be with us indefinitely the opportunity for such treatment should be more general.

EDWIN T. ROLLINS, M.D.

# WOMEN INTERNES AT THE BOSTON CITY HOSPITAL

Editor, Boston Medical and Surgical Journal:

For twenty-five years women physicians have been applying for internships at the Boston City Hospital. During this time there have been women house officers and assistant physicians at the Peter Bent and Massachusetts General Hospitals in Boston and at the municipal hospitals in New York, Chicago, San Francisco and other large cities. There have been one or two women assistant physicians appointed at the Boston City Hospital, but all applications for admission as house officers have been refused. The cause alleged has always been poverty of sleeping quarters, which in a quarter of a century might have been remedied.

The Boston City Hospital is as large as the Massachusetts General Hospital and the Peter Bent Hospital combined. The experience in medical and social welfare gained there cannot be overestimated. The need of women physicians in an institution where there are so many women patients and nurses cannot be overemphasized.

The President of the Massachusetts Medical Society, the Friends of Medical Progress, the deans of both Grade A medical schools admitting women, agree and are ready to cooperate in attempting to convince the Boston City Hospital trustees that the time has come to admit women on the same terms as men to this hospital. A committee from the Boston League of Women Voters is soliciting social and political support, but we believe that if a sufficient number of medical men would petition the trustees no political interference would be needed.

MABEL D. ORBWAY.

## MISCELLANY

MASSACHUSETTS DEPARTMENT OF PUBLIC  
HEALTHDISEASES REPORTED FOR THE WEEK ENDING  
OCTOBER 18, 1924

Disease	No. of Cases	Disease	No. of Cases
Anterior poliomyelitis	10	Ophthalmia neonatorum	21
Chickenpox	110	Pellagra	1
Diphtheria	118	Pneumonia, lobar	54
Dysentery	1	Scarlet fever	146
Epidemic cerebrospinal meningitis	6	Septic sore throat	2
German measles	5	Syphilis	43
Gonorrhea	70	Suppurative conjunctivitis	13
Hookworm	1	Tuberculosis, pulmonary	103
Influenza	2	Tuberculosis, other forms	20
Measles	58	Typhoid fever	16
Mumps	30	Whooping cough	62

## CONNECTICUT DEPARTMENT OF HEALTH

WEEKLY MORBIDITY REPORT FOR THE WEEK ENDING  
OCTOBER 11, 1924(Including all cases reported before 11 A. M., Tuesday,  
October 14, 1924)

Diphtheria	New Haven	1
Fairfield County	New London County	3
Bridgeport	Groton (B)	1
Danbury (C)	Norwich (C)	1
Hartford County	Windham County	1
Berlin	Windham	1
Hartford	State total	13
New Britain	Last week	7
Southington		
Litchfield County	Scarlet Fever	
Torrington (B)	Fairfield County	5
New Haven County	Bridgeport	1
Beacon Falls	Greenwich	1
New Haven	Shelton	1
Wallingford (B)	Stamford (C)	1
Waterbury	Stamford (T)	1
New London County	Hartford County	2
Jewett City	Hartford	1
New London	Manchester	1
Norwich (C)	New Britain	1
Sprague	Southington	1
Stonington	Windsor Locks	1
Waterford	Litchfield County	8
State total	Thomaston	2
Last week	New Haven County	2
The following diphtheria bacilli carriers were reported:	Derby	2
Hartford	Guilford	1
New Haven	Madison	1
Stonington	New Haven	1
Tolland	Seymour	1
Waterbury	New London County	5
	Norwich (C)	4
	Windham County	1
	Putnam (C)	1
	Woodstock	1
	State total	42
	Last week	36
	Measles	
Fairfield County	Fairfield County	1
Bridgeport	Danbury (T)	1
Danbury (C)	Norwalk	1
Norwalk	Hartford County	1
Hartford County	Manchester	1
Hartford	West Hartford	1
Manchester	New Haven County	1
West Hartford	Bristol	1
New Haven County		

New Britain	1	Oxford	3
Suffield	1	Waterbury	2
Litchfield County	1	New London County	4
Torrington (B)	1	New London	6
New Haven County	1	Tolland County	1
Ansonia	2	Willington	46
New Haven	2	State total	19
State total	9	Last week	19
Last week	1	Other Communicable Diseases	
Whooping Cough		Chickenpox	14
Fairfield County	1	Dysentery (bac.)	1
Greenwich	2	Encephalitis epid.	1
Norwalk	1	Mumps	14
Stamford (C)	1	Pneumonia (lobar)	20
Hartford County	1	Poliomyelitis	10
Bristol	1	Tetanus	3
East Hartford	2	Tuberculosis (pul.)	33
Hartford	4	" (other forms)	9
Southington	6	Chancroid	1
New Haven County	14	Gonorrhoea	18
New Haven		Syphilis	22

## CONNECTICUT DEPARTMENT OF HEALTH

WEEKLY MORBIDITY REPORT FOR THE WEEK ENDING  
OCTOBER 18, 1924(Including all cases reported before 11 A. M., Monday,  
October 20, 1924)

<i>Diphtheria</i>		<i>Scarlet Fever</i>	
Fairfield County	3	Fairfield County	3
Bridgeport	2	Bridgeport	1
Danbury (C)	2	Danbury (C)	2
Stamford (C)	3	Greenwich	1
Stratford	2	Shelton	1
Hartford County	—	Stamford (C)	5
Hartford	3	Hartford County	1
New Britain	5	Enfield	1
New Haven County	—	Hartford	6
Ansonia	2	Manchester	1
Derby	1	New Britain	6
Madison	1	Litchfield County	—
Wallingford (B)	2	Litchfield	4
Waterbury	8	Thomaston	3
New London County	—	Middlesex County	—
Norwich (C)	3	Middletown (T)	3
Stonington	2	New Haven County	—
Waterford	1	Ansonia	1
Tolland County	—	Derby	2
Stafford Springs	1	Madison	2
State total	39	Milford	1
Last week	29	New Haven	7
The following diphtheria bacilli carriers were reported:	—	Waterbury	4
Hartford	1	New London County	—
New Haven	5	Colchester	1
Southington	8	New London	1
Stonington	1	Norwich (C)	1
	—	Windham County	—
	—	Putnam (C)	1
	—	State total	56
	—	Last week	42
<i>Typhoid Fever</i>		<i>Measles</i>	
Fairfield County	—	New Haven County	1
Stamford (C)	1	Madison	2
Hartford County	—	New Haven	1
New Britain	2	Waterbury	1
Litchfield County	—	State total	4
Sharon	1	Last week	9
New Haven County	—	Whooping Cough	—
New Haven	—	Fairfield County	—
Wallingford (B)	1	Greenwich	—
State total	6		
Last week	13		



New Canaan	4	Other Communicable	
Stamford (C)	3	Diseases	
Stratford	6		
Hartford County		Cerebrospinal men.	2
Southington	4	Chickenpox	21
Suffield	2	Encephalitis epid.	3
New Haven County		Favus	1
Ansonia	2	German measles	5
Cheshire	1	Influenza	2
Meriden (C)	4	Mumps	9
New Haven	4	Pneumonia (lobar)	17
Oxford	2	Poliomyelitis	2
New London County		Septic sore throat	1
Waterbury	5	Tuberculosis (pul.)	24
New London	5	" (other forms)	2
State total	51	Ophthalmia neo.	1
Last week	46	Gonorrhoea	30
		Syphilis	23

# RHODE ISLAND STATE BOARD OF HEALTH

## CONTAGIOUS DISEASES REPORTED FOR THE WEEK ENDING OCTOBER 4, 1924

<i>Diphtheria</i>		<i>Chickenpox</i>	
Cumberland	1	Burrillville	1
Lincoln	1	Providence	1
Newport	2		
North Kingstown	1	<i>Mumps</i>	
Pawtucket	3	Little Compton	1
Providence	7	Providence	2
<i>Scarlet Fever</i>		<i>Whooping Cough</i>	
Central Falls	1	Providence	4
Cumberland	1		
Providence	1	<i>Measles</i>	
<i>Poliomyelitis</i>		Providence	2
Cranston	1	<i>Typhoid Fever</i>	
East Providence	1	Providence	4

## THE DEDICATION OF THE NEW OUT-PATIENT DEPARTMENT OF THE BOSTON CITY HOSPITAL

On Tuesday, October 21, the friends of the City Hospital gathered in the Assembly Room of this institution for the purpose of participating in the dedicatory exercises of the new Out-patient Department, constructed at a cost of \$630,000.

Dr. Edward N. Libby, Chairman of the Out-patient Staff Committee, presided and, after welcoming the Mayor of Boston, Trustees and friends of the Hospital, spoke as follows:

Your Honor, the Honorable Board of Trustees, Visiting Staff and friends:—

We are met here today for the purpose of dedicating this building. As Chairman of the Staff Committee on Out-Patient affairs it is my duty and privilege to preside at this meeting. The Staff of the Boston City Hospital wish to express their gratitude and appreciation to His Honor, the Mayor, to the Board of Trustees and to our Superintendent, for the vision and forethought which have made possible this magnificent building, with its wonderful equipment. To the men and women who are assembled here today and who have been associated with this Hospital, either in its honorable past or its present, this is a happy event. It is more than an

event,—it marks an epoch in the history of preventive medicine.

Let me point out to you the growth of out-patient departments in the United States. In 1800 there were 3, in 1900 there were 100, in 1910 there were 650, and in 1922 there were 4,000. In them, during 1922, 7,000,000 people were treated, with a total of 30,000,000 visits. These figures demonstrate the great impetus that has been given dispensary work in the past ten years. These out-patient departments are most important factors in carrying out community health programs. Their object is to make readily available to the poor and unfortunate the best facilities for the care of the sick and for the maintenance of health. They have another important function,—the dissemination of knowledge concerning health. In other words, it is their aim to make health contagious instead of disease. It has been stated that the degree of civic virtue of any community may be measured by the care it gives to its sick and unfortunate. If this be true, Boston may well hold up her head among the cities of the world, for nowhere has more wisdom, humanity and vision been exhibited in hospital building devoted to the public service. No one can more fully appreciate nor more fittingly express the aims and ideals of this great institution than one who has spent his entire professional life in its service,—first as Visiting Physician, later as Consulting Physician, and now as a member of the Board of Trustees. It gives me great pleasure to introduce such a man,—Dr. George G. Sears. Dr. Sears took as his theme:

## THE RELATION OF THE OUT-PATIENT DEPARTMENT TO THE HOSPITAL

As a member of the Board of Trustees of the Boston City Hospital, it is my privilege to welcome you to an event which marks another epoch in its history. Founded some sixty years ago to care for the indigent and sick citizens of Boston, it has nobly performed its task and our over-filled wards are an embarrassing testimonial of the appreciation of the public. Since that time the conception of the functions and duties of a hospital has broadened and it is realized that they are not limited to the routine care of bedridden or ambulatory patients and the education of doctors and nurses. It must also be a centre where the causes of disease and the means for its prevention should be sought. In response to this demand, the Thorndike Memorial Building was opened a year ago, in whose well-equipped laboratories the cases of those who come to us in their distress may be studied by all modern methods and the science of medicine be advanced by intensive research conducted by experts.

The dedication of our new Out-Patient Building is a further step in a new medical era. The grim struggle with death over the patient critically ill with pneumonia, when recovery may be certain if life can be prolonged for a few

hours, or the dramatic restoration of one apparently dying of heart disease, drowsy from the effects of his own poisons, almost unrecognizable from dropsy, each breath a laborious process, has too long blinded the physician to the great fact that hospital wards are filled with the evidences of failure. They are the wrecks which might have been saved by preventive medicine or by an early recognition of the meaning of the first symptoms of degenerative disease, for in the millennium for which we are all working, but, alas, without hope of full success, our wards would be reserved to furnish euthanasia to the aged. When this Hospital was founded, the Out-Patient Department was added solely with the idea of serving those who suffered from minor illnesses and who were able to walk to their physician, and a few rooms in the basement of one of the original buildings was deemed sufficient for the purpose. Even now an appointment in this Department is sought by young medical men chiefly as a stepping-stone to service in the wards, where they contemplate the results of failure; failure to educate the public in the factors which make for health and failure to inculcate the necessity to obey its laws; failure because the earliest stages of disease went unrecognized and the transmission of communicable diseases was not prevented, failure, too, in part due to conditions outside of the control of the physician or beyond the compass of attainable knowledge, but in part due to the lack of realization that the greatest advances in medicine may be made by a study of the patient before he has sought asylum in the wards. As the trend of medical practice has tended more and more toward efforts for the prevention and prophylaxis of disease, the rôle which the Out-Patient Department plays has become one of major importance in Hospital administration. It gives opportunities for service to the individual at the time when cure is more easily attained; it acts as a barrier between the ailing patient and the wards; and it furnishes a field for research which has been hardly touched. It requires no great prophetic vision to predict that discoveries will be made there which will be rated among the most important in medicine.

Sir James McKenzie points out in that most notable book, "The Future of Medicine," that we have established great hospitals for the treatment of patients suffering from disease in its advanced stages, and have equipped magnificent institutions for the study of disease after the patient has died, but have given little attention to its detection and cure before it has damaged the tissues, or to the underlying conditions which determine its onset or modify its course. As our knowledge of the clinical effects of disease has been chiefly acquired from a correlation of the clinical phenomena with the pathological changes found at autopsy or at operation this was an inevitable result and the

laboratory has thus assumed an exaggerated importance as the main source of advancement in medicine, and its mechanical appliances, which are chiefly useful after gross changes have occurred, have come to be regarded as an essential means to diagnosis. Our interest in disease has been so stimulated that the interests of the patient have seemed to be forgotten. As Dr. McKenzie says, "few attempts have been made to train men for the detection of the disease when there is a hope of cure. There is, it is true, an impression that the methods of diagnosis are so developed, that disease is now recognized at the earliest stage at which it is humanly possible to recognize it, and that any phenomena that occur earlier are so vague and indefinite, that no clear information can be obtained from them. This view is not justified. There are evidences which would surely indicate the nature of the disease in its earliest stages, were we capable of detecting them: the reason why we fail to detect these evidences is, that medical knowledge has not yet progressed far enough to inform us how to set about finding them." So completely has the idea prevailed that medicine is now best studied in the laboratory that the young medical man who is seeking academic honors is advised that preferment in teaching clinical medicine is most surely gained by devoting himself to the laboratory side. An intimate acquaintance with the test-tube and the microscope is placed above an intimate association with the patient. I would not detract in any way from the discoveries which have emanated from the laboratory during the past forty years; their effect has been revolutionary, but I do complain that they have tended to induce a perverted view of the problems which the practice of medicine presents in the wider experience of the general practitioner. They have aided preventive medicine by discovering the etiology of many of the infectious and occupational diseases; they have made possible the diagnosis of a limited number of affections which would be impossible by purely clinical methods and they are often a distinct and at times a necessary help after gross changes have occurred; but the success of laboratory research has tended to discourage efforts along other lines to discover methods of diagnosis which will reveal the nature of the disease at its inception,—in what might be called the pre-laboratory stages,—before the tissues have been damaged.

For the solution of such problems, medical research must turn from the laboratory to the patient. Though the former may be called upon for help, especially in the chemical field, success will depend not on mechanical appliances but on clinical acumen, on close observation and the trained senses, for the earliest record of disease is written in the appearance and symptoms of the patient. These can be cultivated and acquaintance with the successive phases of disease be obtained only by intimate contact with him. The general practitioner with a large

family clientele would be the ideal person to undertake the task, but modern medicine has been making him an historic figure, and the same qualities which ensure success in this form of research work would limit his opportunities by filling his office with those who are clamoring for immediate relief. The only substitute is an out-patient department which should assume the function of a research laboratory to discover the way to diagnosis before recognizable pathological changes have occurred. Such a conception demands the highest qualities of the physician. The out-patient department should no longer be a place where the young aspirant for a position as visiting physician in the wards impatiently waits for time, by death or resignation, to remove the men who block his path, but one where he may be taught to rely on his God-given faculties in the study of the varying phases of disease from its onset and learn that clinical knowledge is acquired only by long and arduous labor. It is a place where in collaboration with older men, who will find full scope for their wider experience, he may help to solve some of the many medical problems which still confront us, and, as Dr. Rowen emphasized in his address at the opening of the Thorndike Memorial Building, since no hospital can attain its highest usefulness without affiliation with the Medical School, it should give opportunity to the student, under competent instructors, to train himself to serve the public through whose generosity his education has been obtained.

In opening this building, whose construction and splendid equipment were made possible by the generosity of the citizens of Boston, the far-sighted coöperation of the Mayor and the City Council and the expert supervision of our Superintendent, the Trustees confidently expect that the traditions of the past will be maintained, and that in the field of medical research it will prove a worthy rival of the Thorndike Memorial Building.

Dr. Libby then said:

It is not my purpose to detain you with any ideas of my own, but it is impossible for me to forego this opportunity to express again the gratitude of our Staff to the present City Administration and to our Trustees for the splendid building program whose development has been made possible through their efforts. In every generation there are men whose broad vision and deep human sympathy enable them to appreciate that any progress which does not improve the condition of the masses is not true progress. Our Mayor has long been known for his unflinching sympathy with the sick and needy, and his untiring efforts in their behalf. I have the honor and pleasure to introduce the Mayor of the City of Boston—Honorable James M. Curley.

The Mayor expressed his pleasure in meeting the guests of the City Hospital and congratulated

the officials of the Hospital who can now see the realization of their ambitions after long and vexatious delay.

He paid a merited tribute to the architects and builders for their devotion to the problems involved and extolled the spirit of the citizens of Boston which led to the appropriation of funds which are always justifiable when applied to human needs.

He outlined plans under consideration for the further care of the sick and needy in Boston and referred to the diminishing number of deaths from tuberculosis which he expects will be below four hundred per year within a few years.

Speaking of the health units now being developed in the City, he felt that by a large expenditure of money much greater progress will be made in preventing sickness and promoting health and that the great outstanding purpose at the present time is to coöperate all the health agencies in order to secure the greatest efficiency and eliminate duplication of effort.

The great danger of civic life is, he felt, stagnation, but that Boston shows no tendency along that line for the spirit of the City is service and being of service.

After the formal exercises were finished the new building was thrown open for inspection.

#### RESULTS OF THE EXAMINATION CONDUCTED BY THE MASSACHUSETTS BOARD OF REGIS- TRATION IN MEDICINE, SEPTEMBER, 1924

##### REGISTERED

Bray, Thomas Ambrose, Holliston, Mass.  
Byers, Randolph Kunhardt, 277 Adams Street, Milton, Mass.  
Camp, John Dexter, Massachusetts General Hospital, Boston, Mass.  
Cariani, Mario John, 879 Worthington Street, Springfield, Mass.  
Cheetham, Donald Butterworth, Worcester State Hospital, Worcester, Mass.  
Conroy, Augustine Edward, 29 Essex Street, Andover, Mass.  
Daley, Joseph Arthur, 14 Broards Avenue, South Natick, Mass.  
Donovan, Arthur Bland, Boston City Hospital, Boston, Mass.  
Dube, Isidore Peter, Chelsea Memorial Hospital, Chelsea, Mass.  
Elliott, Blanche, 83 Monroe Street, Haverhill, Mass.  
Fraser, Leopold Hury, 39 Harbor Street, Salem, Mass.  
Haagensen, Cushman Davis, Boston City Hospital, Boston, Mass.  
Howard, Louis Guilford, 56 Bellevue Street, West Roxbury, Mass.  
Johnson, William Joseph, 55 Oakland Street, Lowell, Mass.  
Kalish, Louis Leo, 94 Nightingale Street, Dorchester, Mass.  
Kelley, Julius Goddard, P. O. Box 91, Dennis Port, Mass.  
LeFurgy, William Godfrey, 1677 Commonwealth Avenue, Boston, Mass.  
Macdonald, Thomas Henry, Box 202, New Glasgow, N. S.  
McCarthy, Walter Rochefort, 38 Exeter Street, Lawrence, Mass.

McNamara, John Joseph, 494 Gorham Street, Lowell, Mass.  
 Merliss, Eugene, 6 Segal Street, Roxbury, Mass.  
 Mills, Ashley Ernest, Lawrence General Hospital, Lawrence, Mass.  
 Moulton, Lillian Gertrude, Box 50, Hathorne, Mass.  
 Perkins, Beatrice Louise, West Lebanon, Me.  
 Pickwick, Erskine Richards, Newton Hospital, Newton Lower Falls, Mass.  
 Rooney, Paul Neill Anthony, 20 Fenwood Road, Boston, Mass.  
 Silver, Maurice Jacob, 536 Commonwealth Avenue, Boston, Mass.  
 Simmons, Arthur Maxwell, Cambridge City Hospital, Cambridge, Mass.  
 Snelerson, Hyman, Hospital Station, Binghamton, N. Y.  
 Spellman, John William, Massachusetts General Hospital, Boston, Mass.  
 Steele, Fred Elton, Jr., 600 Washington Street, Boston, Mass.  
 Walsh, John Sylvester, Carney Hospital, Longwood Avenue, Boston, Mass.

#### APPLICANTS REJECTED IN SEPTEMBER EXAMINATION AND YEAR OF GRADUATION

Middlesex, 1923-23-23-23-23-23-24.  
 Physicians and Surgeons, Boston, 1923.  
 St. Louis College of Physicians and Surgeons, 1923-24-24-24-24-24.  
 Massachusetts College of Osteopathy, 1923-23-24-24-24.  
 American School of Osteopathy, 1924.  
 Jefferson, 1920.  
 Naples University, 1921.  
 Tufts, 1924.  
 University of Oporto, 1919.  
 University St. Vladimir, 1919.  
 University of Vienna, 1923.  
 Imperial Nicholas University, 1914.  
 Vanderbilt, 1923.  
 Athens University, 1922.

#### REGISTERED OCTOBER 23, 1924 (SPECIAL EXAMINATION)

Hamilton, Bengt. Knutson, Children's Hospital, 300 Longwood Avenue, Boston, Mass.  
 Morrison, Sidney Leon, 267 Pearl Street, Burlington, Vt.  
 Tanguay, Joseph Edgar, 172 Main Street, Woonsocket, R. I.

#### GOLDEN RULE SUNDAY

DECEMBER seventh has been set aside as Golden Rule Sunday by the Golden Rule Co-operating Committee of the Near East Relief, the object being to promote the spirit of good fellowship and peace among the nations of the world, and to provide sustenance for the 40,000 orphans under American care in the Near East. In observation of this day we are asked to eat such a simple meal as is served each day to the children in Near East Relief orphanages in Syria, Palestine, Greece and the Russian Caucasus and to make a thank offering so that the children may continue under our protection until they are ready for self-support.

Altogether the Near East Relief has cared for over 100,000 children, and during the past year 14,000 were absorbed in industries or placed with families. Conditions have already so improved that the orphanage death rate is now lower than that of the average American city. A successful battle has been waged

against trachoma, and 33 hospitals and 61 clinics are maintained with an average of 395,427 treatments per month. The work is supervised by 8 American doctors, 21 American nurses, 52 local doctors and 121 native nurses.

#### NATIONAL BOARD OF MEDICAL EXAMINERS

SURGEON GENERAL MERRITT W. IRELAND, President of the National Board of Medical Examiners, whose headquarters are in Philadelphia, has announced the names of the candidates who achieved the highest honors in the Board's summer examination of students in Class A Medical Colleges throughout the country for qualifying them to enter the practice of medicine.

In the examination given to students at the end of the second year of their course, the 10 candidates receiving the highest ratings out of the 238 examined were:

Theodore Dunham, Jr. of New York, who had his medical training at Cornell University Medical College; Henry S. F. Cooper of Coopers-town, N. Y., Harvard University Medical School; Mary Agnes Jennings of New York, Columbia University College of Physicians and Surgeons; Walter Francis Duggan of Monson, Mass., Columbia University College of Physicians and Surgeons; William Henry Crawford of Reynoldsville, Pa., University of Pennsylvania School of Medicine; Rebecca B. Carter of Washington, Conn., Cornell University Medical College; William Borden Stevens of Newport, R. I., Harvard University Medical School; Willard Owen Thompson of Halifax, Nova Scotia, Harvard University Medical School; Elmore Russell Bailey of Chicago, Ill., Rush Medical College, and Herman Slass of Jamaica, L. I., Columbia University College of Physicians and Surgeons.

Mr. Dunham who earned the highest number of credits had 402.1 out of a possible 425. He is a member of the Class of 1925 at Cornell. Dr. Cooper, the second, had 388.8 out of 425 credits.

The ten highest candidates of the 180 who took the examination for students completing their third and fourth years were:

Dr. Jean Crump of Pittsburgh, Pa., who had her medical training at the Woman's Medical College of Pennsylvania; Herbert Monheimer of Smethport, Pa., University of Pennsylvania School of Medicine; Jacob Morton Mora of Chicago, Ill., University of Illinois College of Medicine; Carl E. Bachman of Reading, Pa., University of Pennsylvania School of Medicine; Martha C. Souter of Whitehall, N. Y., Cornell University Medical College; Kenneth E. Appel of Lancaster, Pa., Harvard University Medical School; J. Henry Rieniets of Arlington, Iowa, State University of Iowa College of Medicine; Evelyn Holt of Summit, N. J., Cornell University Medical College; Herman A. Lawson of

Newport, R. I., Harvard University Medical School, and Charlotte McCarthy of Evanston, Ill., Rush Medical College.

#### A NEW PORTABLE X-RAY OUTFIT

A PORTABLE X-ray outfit, which weighs only 20 pounds and which can be carried with ease in a convenient carrying-case, has been developed by Dr. W. D. Coolidge and his associates in the research laboratory of the General Electric Company at Schenectady. A very small X-ray tube operates in the same oil and in the same metallic container with a high tension transformer.

The resulting X-ray generating unit, with its compactness, ease of manipulation, and freedom from external electrostatic field, seems especially well adapted for numerous lines of physical investigation work, and, in a slightly different mounting, for the physician and dentist. Many of the sets of the latter type have been used by dentists and physicians in the past year.

This set does not, however, replace the portable X-ray outfit developed during the war for use in field hospitals and later made available for physicians to use at the bedside of patients. It has a different field for usefulness.

To use the new apparatus it is merely necessary to connect an ordinary extension cord in the nearest socket of the house circuit. X-rays are then produced merely by pressing a switch. Since the outfit is totally encased in a metal container, which in turn is within a leather-covered, wooden carrying-case, there is no high voltage hazard. The outfit is grounded to the container.

#### BEVERLY HOSPITAL

A DEMONSTRATION clinical meeting was held at the Beverly Hospital, Tuesday, October 21, at 4.00 P. M. Interesting cases were shown and opened for discussion.

JAMES A. SHATSWELL, M. D., *Pres.*  
RALPH E. STONE, M. D., *Sec.*

#### AUTOMOBILE FATALITIES

THE Department of Commerce has published the record of automobile fatalities in the Registration Area.

Although the record is disconcerting, it is not complete, for many fatalities in which automobiles figure are credited to other statistics. Grade crossing fatalities are applied to railroads, for example.

California had the highest rate per 100,000 population for 1923, amounting to 32.6. The Massachusetts rate was 15.2, which compares very favorably with the larger states.

A resume of the figures is as follows:

The returns show that during the year 1923,

14,412 deaths resulting from accidents caused by automobiles and other motor vehicles (excluding motorcycles) occurred within the death registration area of the United States (exclusive of Hawaii), which area contains 87.6 per cent of the total population. This number represents a death rate of 14.9 per 100,000 population as against 12.5 in 1922, 11.5 in 1921, 10.4 in 1920, 9.4 in 1919, and 9.3 in 1918. In the 30 states for which data for 1918 are available, the actual number of these deaths increased from 7,255 in that year to 13,043 in 1923, the corresponding rates for these two years being 9.2 and 15.4.

#### BOSTON TUBERCULOSIS ASSOCIATION BULLETIN NO. VI

##### PHLYCTENULAR KERATITIS

BY GEORGE S. DERBY, M. D.

*Ophthalmic Chief of Service,*

Massachusetts Charitable Eye and Ear Infirmary, Boston.

##### INTRODUCTORY NOTE BY JOHN B. HAWES, 2ND, M. D., PRES., B. T. A.

THE following bulletin by such an authority on eye conditions as Dr. George S. Derby should be read by every physician. Out-Patient Departments and dispensaries are crowded with children of the type to which he refers, who come for treatment of their eyes and in far too many instances it is the eye alone that receives any attention. Dr. Derby, who has done pioneer work in this direction, recently asked the Boston Tuberculosis Association to help in handling this problem. It is as he states not by treatment of the eyes but by general hygienic measures, by introducing fresh air, sunshine, good food and right habits of living into the homes of these children that the disease in the eye which may later lead to serious blindness is to be checked.

Our Association, therefore, has provided a trained worker who is to visit such children in their homes and to teach them so to live that the damage done by this form of tuberculosis may be reduced to a minimum.

##### PHLYCTENULAR KERATITIS

Poor vision begets incompetence. Poor vision due to scarring of the cornea in children and young adults is often caused by Phlyctenular Keratitis, which therefore becomes a problem of great importance.

Phlyctenular Keratitis blossoms from the soil of under-nutrition, anaemia and underweight, and it usually accompanies Juvenile Tuberculosis, most frequently of the skin of the face and mucous membranes of the nose, less often of the bones, lymph glands, lungs, tonsils and adenoids, and nasal sinuses. While the tubercle bac-



illus has never been found in the lesions of the eye, much clinical and experimental evidence has been accumulated to indicate that Phlyctenular Keratitis is a sequel of Tuberculosis.

The eye lesions, or phlyctenules, usually multiple, running an acute course of one to two weeks, by predilection seeks the limbus but also occur on the cornea and conjunctiva. The phlyctenule, red at the limbus, grey on the cornea, consists of a nodular collection of lymphocytes just beneath the epithelium, mounding up the epithelium overlying it, and accompanied by an injection of the adjacent conjunctival vessels. The phlyctenule may absorb without complications, or it may become transformed into a superficial ulcer through the loss of epithelium covering it. An ulcer, once formed, may either heal without scar, if superficial, or heal with scar, the density of which depends upon the depth to which the ulcer penetrated, or it may go on to perforation of the cornea. A persistent vascularization of the cornea (pannus) from the adjacent conjunctival vessels can usually be found at the site of the deeper ulcers.

Profuse lacerimation appears early, accompanied by marked photophobia, which causes the patient to shield the eye from light through burying the head in the hands or bedclothes. Attempts to examine the eye are strenuously resisted, and lid spasm is pronounced, during the acute attack.

The prognosis depends upon the location and extent of the scars, especially those immediately overlying the pupil. Unless the general health is improved, there is a marked tendency to recurrences, which sooner or later will usually scar the pupillary area. While the eye is rarely made entirely blind, sufficient impairment of sight is produced to cause the child to fall behind in school. Occasionally, the poor vision may uncover latent tendency to squint.

Manifestly, the most urgent treatment is prevention, early attention to child health offering a fruitful field, to reduce the incidence and damage from the affection. The acute exacerbations of the disease require constitutional treatment as for any other form of tuberculosis, and local treatment designed to put the eye at rest, and minimize complications.

#### NEWS ITEM

##### APPOINTMENTS AT THE HARVARD MEDICAL SCHOOL

THE following appointments were consented to at a meeting of the Board of Overseers of Harvard University on October 20, 1924.

William Lorenzo Moss, M. D., Assistant Professor of Bacteriology, for one year from September 1, 1924.

Calvin Barstow Faunce, Jr., M. D., and Philip Hammond, M. D., Instructors in Otolaryngology, for three years from September 1, 1924.

Robert Bayley Osgood, M. D., was elected the John B. and Buckminster Brown Professor of Orthopaedic Surgery to serve from September 1, 1924. (Formerly Professor of Orthopaedic Surgery.)

#### REMOVALS

DR. EDWARD J. MAHONEY's office is now at 73 Chestnut St., Springfield.

DR. BENNETT SOLOMAN has returned to Springfield and has his office at 1329 North St.

DR. HUNTER ROBB has been transferred from the Middlesex East to the Suffolk District. His present address is 16 West Cedar St., Boston.

DR. MARY WRIGHT of Newton Center now has an office at 21 Bay State Road, Boston.

DR. FRANK HOLYOKE has moved his office from 280 High to 187 Walnut St., Holyoke.

DR. CARL W. ROSENBLUM's office is now 419 High St., Holyoke.

DR. MICHAEL C. MCGINLEY has moved from Ipswich (Essex South) to Lynn (same district). His office is at 67 Nahant St.

DR. W. B. ADAMS of Springfield now has his office at 174 State St.

DR. RAYMOND L. BARRETT now has his office at 530 Summer Ave., Springfield.

DR. JOHN M. BIRNIE now has his office at 14 Chestnut St., Springfield.

DR. JOSEPH N. BOYER of Springfield has moved his office from 175 State St. to 70 Fort Pleasant Ave.

DR. F. F. DEXTER has moved his office from 175 State St. to 6 Chestnut St., Springfield.

DR. ALEXANDER C. EASTMAN has moved his office from No. 6 to No. 14 Chestnut St., Springfield.

DR. ALFRED M. GLICKMAN has moved his office from 476 Chestnut to 136 Main St., Springfield.

DR. FREDERICK E. HOPKINS has moved his office to 146 Chestnut St., Springfield.

DR. LEONARD C. DURSTOFF of Chelmsford now has an office at 8 Merrimack Sq., Lowell.

DR. ARCHIBALD R. GARDNER has moved his office from 18 to 16 Shattuck St., Lowell.

DR. ABBY N. LITTLE has moved from Newburyport (Essex North) to the School for the Feeble Minded at Laconia, N. H. (Non-Resident List.)

DR. ROBERT N. NYE has changed his address in Brookline to 26 Lawrence Rd.

DR. FRANCIS D. RANDALL is now at 12 Maplewood Street, Malden.

DR. WILLIAM P. MURPHY of Jamaica Plain now has an office at 311 Beacon Street, Boston.

DR. A. WILLIAM REGGIO has moved from Beacon Street to 374 Marlborough Street, Boston.

#### RECENT DEATHS

DR. WALTER CLEMENT KENNEY, a Fellow of The Massachusetts Medical Society, died at his home in Winchendon, October 14, 1924, at the age of 45.

He was a graduate of Tufts College Medical School in 1904 and joined The State Medical Society from Tewksbury the following year. He moved to Winchendon in 1909.

DR. IRA HUMPHREY PROUTY, of Keene, N. H., died at Hartford, Conn., October 8, 1924, at the age of 39. He was the son of Dr. Ira J. Prouty, a prominent physician of Keene, a graduate of the Johns Hopkins University Medical Department, in 1911, made a specialty of pediatrics and at the time of his death was serving as chief of the laboratory department of the Travelers Insurance Company, at Hartford. Besides his widow, who was Miss Jennie H. Jordan of Somerville, he leaves a son and a sister.

#### NOTICES

##### MASSACHUSETTS HOMEOPATHIC MEDICAL SOCIETY

THE Fall meeting of the Massachusetts Homeopathic Medical Society will be held at the Homeopathic Hospital on Wednesday, November 5th.

##### CENSORS' MEETING

THE Censors of the Suffolk District Medical Society will meet for the examination of candidates at the Medical Library, No. 8 The Fenway, Thursday, November 6, 1924, at 4.00 o'clock.

Candidates should make personal application to the Secretary, and present their medical diploma at least one week before the examination.

LESLEY H. SPOONER,  
*Secretary.*

520 Commonwealth Ave.

#### THE TRUDEAU SOCIETY

THE second meeting of the Trudeau Society of Boston will be held at the Boston Medical Library on Thursday, October 30, at 8.15 P. M. Dr. John B. Hawes, 2d, President of the Boston Tuberculosis Association, and Dr. Eli Friedman, Camp Physician, will report on the studies of bronchial gland tuberculosis at Prendergast Preventorium.

##### STAFF CLINICAL MEETING, BOSTON CITY HOSPITAL, CHEEVER SURGICAL AMPHITHEATRE, FRIDAY, NOV. 7, 1924, AT 8.15 P. M.

Confusing Vaginal Tumors—N. R. Mason.  
Evolution and Treatment of Complete Proidentia—R. M. Green.

Pyelography in Pyelitis of Pregnancy—J. T. Williams.

Cesarean Section, Its Use and Abuse—F. L. Good.

Vomiting of Pregnancy—J. P. Cohen.

Painless Childbirth—H. V. Hyde.

Chorioepithelioma Malignum—F. J. Lynch.

Sole Printing of Newborn Babies—R. D. Margeson.

Open Discussion, physicians, medical students and nurses invited.

Refreshments.

JOHN J. DOWLING, *Superintendent.*

##### APPOINTMENT OF DR. W. J. V. OSTERHOUT

THE Rockefeller Institute for Medical Research announces the appointment to the Scientific Staff of Dr. W. J. V. Osterhout, Professor of Botany at Harvard University.

Dr. Osterhout was born in Brooklyn, New York, in 1871 and graduated from Brown University in 1893. He remained at Brown for two years as Instructor in Botany, taking his A. M. there in 1894, meanwhile teaching at the Marine Biological Laboratory at Woods Hole, Mass., in the summers of '94 and '95. In 1895-96 he studied in the laboratory of Prof. E. Strasburger in Bonn, Germany. In 1896 he went to the University of California as Instructor in Botany, remaining there for thirteen years, becoming successively Assistant Professor (1901), and Associate Professor in Botany (1907), which title he held until 1909, when he left California to accept an Assistant Professorship in the Department of Botany at Harvard University, later, in 1913, becoming full Professor. Dr. Osterhout has also served on the following lectureships: Hitchcock Lecturer at the University of California, 1919; Colver Lecturer at Brown University, 1922, and Lowell Lecturer at the Lowell Institute, Boston, 1922-23.

His research has dealt with certain fundamental life processes, including measurements and

mathematical analyses of injury, recovery, and death of cells, and a theory whereby these phenomena may be predicted. His latest work, carried out in Bermuda, consists in extending these conceptions through the study of a multinucleate cell (Valonia) whose extraordinary size permits observations of a kind never before attempted.

These investigations have resulted in certain new points of view, some of which have a bearing on theories of disease.

### LECTURE ON RECOVERY PROCESS AFTER MUSCULAR ACTIVITY

ARCHIBALD VIVIAN HILL, A. M., Sc. D., F. R. S., Professor of Physiology, University College, London, will speak on "Recovery Process after Muscular Activity" at five o'clock on November 21 in Amphitheatre C, Harvard Medical School. Physicians are cordially invited to attend.

It is of interest to note that Dr. Hill was the winner of the Nobel Prize in Physiology last year. Herter Lecturer.

### BRISTOL SOUTH DISTRICT

The program for the Nov. 6, 1924, meeting is as follows:

Preventive Obstetrics, by Dr. Robt. L. DeNormandie.

Following this paper Dr. DeNormandie will give a talk relating to the Joint Committee on Maternal Welfare.

### UNITED STATES CIVIL SERVICE EXAMINATION

The United States Civil Service Commission announces the following open competitive examination:

MEDICAL OFFICER, JUNIOR GRADE—MEDICAL OFFICER, GRADE A—MEDICAL OFFICER, GRADE B

Applications for the positions of medical officer, junior grade, medical officer, Grade A, and medical officer, Grade B, will be rated as received until December 30. The examinations are to fill vacancies in the Indian Service, the Coast and Geodetic Survey, the Panama Canal Service, the Public Health Service, and the Veterans Bureau, at entrance salaries ranging from \$1680 to \$3000 a year.

Appointees to position in the Indian Service will be allowed quarters, heat and light free of cost. Appointees to positions in the Coast and Geodetic Survey are allowed \$2 a day for subsistence in addition to the basic salary.

The eligibles resulting from these examinations will be placed on registers and certified according to their qualifications in the following branches: General medicine and surgery (junior and A grades only), tuberculosis, neuropsychiatry and psychiatry, bacteriology (advanced), pathology, epidemiology, Public Health practice, industrial medicine and hygiene, and child hygiene.

Competitors will be rated on their education, training and experience.

Full information and application blanks may be obtained from the United States Civil Service Commission, Washington, D. C., or the secretary of the Board of United States Civil Service Examiners at the postoffice or custom house in any city.

### SOCIETY MEETINGS

#### Bristol South Medical Society

Next meeting will be held Thursday, November 14, 1924.

#### Essex North District Medical Society

January 7, 1925. Semi-annual meeting at Haverhill.  
May 6, 1925. Annual meeting at Lawrence.

#### Hamden District Medical Society

Meetings to be held on the third Tuesday of January and the third Tuesday in April.  
Dr. William J. Mayo is expected to attend the combined meeting of the four western Districts to be held in Springfield in October.

#### Hampshire District Medical Society

The meetings will be held the second Wednesday of November.  
January, March and May.  
Dr. Channing Simmons will deliver an address at the November meeting. Title: "The Treatment of Cancer, with Especial Reference to Radiation."

#### Middlesex East District Medical Society

Wednesday, November 13. Harvard Club. Dr. George K. Pratt, "Community Aspects of Psychiatry."  
Wednesday, January 21. Harvard Club. Dr. Franklin K. White, "Diagnosis of Gall-Bladder Disease."  
Wednesday, March 13. Harvard Club. Dr. John H. Cunningham, "Urinary Retention: Its Significance and Treatment."  
Wednesday, April 16. Harvard Club.  
Wednesday, May 13. Colonial Inn, North Reading.

#### Middlesex North District Medical Society

January 28, 1925.  
April 29, 1925.

#### Middlesex South District Medical Society

Winter Schedule—The plans for winter meetings of the Society include the stated meetings in October and April, two hospital meetings, and five meetings to be held in conjunction with the Suffolk District Medical Society and the Boston Medical Library (two surgical, two medical, and one general).

#### Norfolk District Medical Society

November 26, 1924. Masonic Temple, Roxbury. Subject: Diabetes—with special reference to insulin. Speakers: Dr. Joslin and probably one other.  
January 27, 1925. Masonic Temple. Subject: "Some Trends of Medical Teaching and Medical Practice." Speakers: Drs. A. S. Egge and W. F. Bowers.  
February 24, 1925. Masonic Temple. Subject: "The Need of Periodical Physical Examinations and How to Make Them." Speaker: Dr. Francis H. McCrudden. A second speaker will be selected to present another subject at this meeting.  
March 21, 1925. Tufts College Medical School. This meeting given over to Drs. Leary and Watters for the purpose of giving us a medical examiners' talk.

#### Norfolk South District Medical Society

Meetings will be held the first Thursday of each month from October to May, inclusive, at 12 noon, at the Norfolk County Hospital, South Braintree.

#### Suffolk District Medical Society

November 12. Surgical Section, in association with the Middlesex South District Medical Society. "The Treatment of Septicemia by the Newer Methods." Dr. Edward B. Pieper, Philadelphia, Pa.  
December 17. Medical Section, in association with the Middlesex South District Medical Society. "The Newer Drug Treatment of Heart Disease." Dr. Paul D. White.  
January 28. General meeting, in association with the Boston Medical Library and the Middlesex South District Medical Society. "Some Experiences of a Medico-legal Pathologist" (lancet slides). Dr. George E. Magrath.  
February 25. Surgical Section, in association with the Middlesex South District Medical Society. "Pyelonephritis." Dr. Arthur H. Crooble.  
March 25. Medical Section, in association with the Middlesex South District Medical Society. "The Treatment of Pneumonia." Dr. Edwin A. Locke.  
April 19. Annual meeting. "Hypertension and Longevity." Dr. Harold M. Frost.

#### Worcester District Medical Society

November 12, 1924. Grafton State Hospital. Dr. Charles Macfie Campbell of the Psychopathic Hospital, Boston, will speak on "Nervous Disorders in Children."  
December 19, 1924. Worcester State Hospital. Subject and speaker to be announced.  
January 7, 1925. Surgical meeting. Place, subject and speaker to be announced.  
February 11, 1925. Memorial Hospital, Worcester. Papers will be read by the members of the hospital staff.  
March 11, 1925. St. Vincent's Hospital, Worcester. Papers will be read by the members of the hospital staff.  
April 9, 1925. Subject and speaker to be announced.  
May 14, 1925. Annual meeting.